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REGULATION OF VELOCITY IN RAPID VOLUNTARY MOVEMENTS

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The undersigned certify that they have read, and
recommend to the Faculty of Graduate Studies and Research,
for acceptance, a thesis entitled REGULATION OF VELOCITY IN
RAPID VOLUNTARY MOVEMENTS submitted by Ted Milner in partial
fulfilment of the requirements for the degree of Doctor of
Philosophy.

Abstract

Regulation of velocity and amplitude in 'ballistic' reciprocating movements and regulation of velocity in 'ballistic' flexion movements of the interphalangeal joint of the thumb were investigated by examining movement trajectories and patterns of activity in the extensor pollicis longus (EPL) and flexor pollicis longus (FPL) muscles.

Subjects performed three tasks involving regulation of movement amplitude and/or peak flexion velocity of reciprocating movements. When a constraint was imposed on only one of these parameters there were always strong linear correlations between peak extension velocity, peak flexion velocity and movement amplitude.

It was not possible to increase the slope of peak flexion velocity versus movement amplitude by keeping amplitude fixed, but the slope was reduced when velocity was kept fixed. Attempting to disrupt the prefered strategy in this way, led to an increase in movement variability. Constraints imposed on peak flexion velocity did not affect peak extension velocity, indicating that the two phases of reciprocating movements could be separately regulated.

Reciprocating movements which were scaled in amplitude also tended to be scaled in time. This scaling was more adequately described in terms of muscle activation patterns than by step shifts in the mechanical properties of the antagonist muscles.

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When reciprocating movements were linked together in a rhythmic sequence the relationship between peak velocity and movement amplitude remained relatively unaltered over a range of tempos, even though movement time shortened as frequency increased. Maintenance of the slope of this relationship was achieved by shortening the duration and increasing the amplitude of e.m.g. bursts in both the EPL and FPL muscles.

Although velocity was regulated with considerable accuracy in 'ballistic' flexion movements, it was apparently not sensed with the same precision. Consistent velocity errors were made when subjects attempted to match the peak velocities under conditions in which the relationship between muscle activity and joint acceleration had been altered, e.g. increasing the angle from which movement was initiated or altering the load.

It seems that rather than relying on afferent feedback from peripheral sensory receptors for information about velocity during 'ballistic' movments, subjects are more likely to base their judgment of velocity on sensations evoked by the voluntary motor command.

Acknowledgments

I am deeply indebted to Robert Rolf who became a part of this project in its early stages, helping me design and build electronic hardware, writing software for averaging, I/O and displays, and acting as my programming consultant. Without him things would have progressed much more slowly. Thanks a million Rob!

Wayne Aldridge and Peter Murphy will not be soon forgotten. In reading this thesis they may smile, for I am certain that here and there they will identify thoughts which they have heard before. I am grateful that neither of them was a firm believer in the orthodox doctrines of neurophysiology. They took an undeserved interest in my work, listened patiently to heretical ideas and far-fetched theories and tried to give me a sense of accomplishment.

Finally, I bid farewell to Dick Stein with the completion of this thesis. He had enough faith in me to let me pursue my own ideas, even though I was often so vague about them that he must have wondered whether I knew anything about motor control at all. His insights were always far superior to mine making me reluctant to speculate in his presence for fear of letting my ignorance show. He has been patient with me and I have learned much from him. I have enjoyed working under his supervision. I will be a member of 'Stein's lab' in spirit, for a long time to come.

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I. INTRODUCTION

A. Rapid Voluntary Movements

Voluntary limb movements about single joints can be broadly classified according to their speed and the patterns of muscle activity associated with them. Stetson and McDill (1923) described three categories based on speed: fixation, slow movements and rapid movements. Fixation, refers to movements of almost imperceptible amplitude which occur when a limb is maintained in a fixed position. Slow movements consist of movements in which a limb is actively displaced as the result of continuous activation of agonist muscles which may be accompanied by varying amounts of activity in antagonist muscles (Wachholder and Altenburger, 1926; Lestienne and Bouisset, 1968). Included in this category are 'smooth' movements (Hallett et al., 1975a) and 'ramp' movements (Desmedt and Godaux, 1978a). In contrast to slow movements, rapid movements, which are impulsive in nature, are characterized by discrete bursts of activity in agonists and antagonists (Wachholder and Altenburger, 1926; Lestienne and Bouisset, 1968; Hallett et al., 1975).

The pattern of activity seen in agonist and antagonist muscles during rapid movements is particularly dependent upon the nature of the braking process. Rapid movements are generally initiated by a burst of activity in the agonist muscles which imparts an accelerative impulse torque to the limb. If this impulse torque is sufficiently small,

visco-elastic forces developed by the joint can provide
enough decelerative torque to brake movement of the limb. No
increase in antagonist muscle activity is required unless it
is desired to decelerate more quickly. To limit movement
amplitude, antagonist muscles must actively participate in
generating decelerative torque (Lestienne, 1979; Hoffman and
Strick, 1982; Marsden et al., 1982). When the antagonists
are being used in this way, the overall pattern of muscle
activity is either biphasic or triphasic, i.e. a burst of
activity in the agonists followed by a burst in the
antagonists, usually followed by a second burst in the
agonists. This reciprocal pattern of muscle activation was
first described by Wachholder and Altenburger (1926). It is
a characteristic feature of rapid limb movements in which
the momentum is too great to be overcome by passive forces
alone.

However, when braking is provided by the action of a
mechanical stop, there is no need for any active
participation by the antagonists. In this case, the movement
can be executed by means of a single burst of activity in
the agonist muscles (Waters and Strick, 1981; Marsden et
al., 1983).

Considerable attention has been focused on rapid
movements recently, because they have characteristics which
suggest that they are preprogrammed by the central nervous
system. In order to be preprogrammed, a representation or
program for a desired movement must exist in the central

nervous system prior to initiation of the movement. During execution of the movement this program can presumably be observed as a patterned sequence of activity distributed among neurons throughout movement-related areas of the brain which interact to generate descending command signals to motoneurons. For a movement to be truly preprogrammed, this activity must be organized in such a way that the desired trajectory can be achieved without the need for error-correcting feedback loops. Thus, a preprogrammed movement is one which could be executed by the central nervous system without reliance on sensory feedback from the periphery.

One reason why rapid movements are believed to be preprogrammed is because they are carried out in such a short space of time that it is doubtful whether even the fastest feedback loops from peripheral sensory receptors could provide effective error correction. Even the fastest reflex pathways require at least 60 milliseconds to effect a correcting response to a disturbance of limb trajectory (Crago et al., 1976; Carlton, 1983). This delay is substantial for a movement which lasts only 100-200 milliseconds. Unless deviations from the desired trajectory could be predicted before their occurrence, by the time they were detected it would be too late to correct them. Furthermore, because of the impulsive nature of rapid movements, any correcting impulse would have to occur early in the movement in order to be effective. It is, therefore,

unlikely that any provision is made during rapid movements for effective error correction.

Keele (1968) defined motor program as "a set of muscle commands that are structured before a movement sequence begins, and that allow the entire sequence to be carried out uninfluenced by peripheral feedback." While this may be an operational definition, it is certainly not physiological. Taken literally, its range of application would be restricted to situations in which peripheral feedback had been eliminated either pathologically or as the result of surgical intervention. Otherwise, it would imply that peripheral reflex pathways are gated off during the execution of the motor program. Even the fastest movements, which appear least susceptible to the influence of afferent activity originating from peripheral sensory receptors, are never completely insensitive to unexpected loading or unloading, as Hallett and Marsden (1979) have demonstrated.

The position taken by Desmedt and Godaux (1978a) is less extreme. They distinguish between movements which are continuously controlled in feedback servo fashion using sensory inputs from the periphery and movements that are triggered as units which must run their full course "without the possibility of modification." The latter are termed preprogrammed or ballistic movements. This is more in line with the view presented above, that any error-correcting command which could be issued to the motoneurons following movement onset would be too late to be effective in altering

the trajectory of a preprogrammed movement.

The strongest support for preprogramming of rapid movements comes from observations of human subjects with neuropathies which have resulted in severe peripheral sensory loss (Hallett et al., 1975a; Rothwell et al., 1982). These subjects were able to execute rapid target-directed limb movements quite accurately while exhibiting a normal pattern of agonist/antagonist muscle activation.

Although under these circumstances appropriate motor activity for rapid movements was generated in the absence of peripheral sensory activity, this does not preclude the possibility that sensory feedback plays a role when sensation is intact. Yet, it appears that the short duration of rapid movements and the relatively long delay between movement onset and subsequent reflex actions of peripheral sensory receptors make it unlikely that peripheral afferent activity can significantly influence the initial burst of activity in the agonist muscles. In addition, the efficacy of the myotatic reflex is depressed in both agonist and antagonist muscles during the acceleration phase of rapid movements (Gottlieb and Agarwal, 1980; Soechting et al., 1981), even further limiting any potential role for sensory feedback. However, during braking the myotatic reflex shows enhanced sensitivity, suggesting that the antagonist burst is reinforced by peripheral afferent activity (Soechting et al., 1981). This is supported by observations of deafferented monkeys and humans (Terzuolo et al., 1974;

Forget and Lamarre, 1982; Jennings and Sanes, 1982) and cats subjected to unexpected limb displacements (Ghez and Martin, 1982). It is possible, therefore, that peripheral sensory feedback is suppressed when its effectiveness is minimal and enhanced only when it can be used to advantage.

B. Movement Control Theories

Two theories on the nature of the control process during rapid positioning movements have come into prominence recently. The first, which will be referred to as the equilibrium point theory, originated with Asatryan and Fel'dman (1965) and has been subsequently elaborated by Fel'dman (1966a, 1966b, 1974a, 1974b, 1980a, 1980b). The second is known as the impulse-variability theory. It was originally proposed by Schmidt et al. (1979) and has since been modified by Meyer et al. (1982). Wallace (1981) related kinematic aspects of the theory to muscle activity.

Equilibrium Point Theory

In Fel'dman's equilibrium point theory, an analogy is drawn between muscles and springs. The active generation of muscle force is described in terms of the same parameters used to characterize a spring, namely, stiffness and zero length. Zero length can be defined in a straightforward manner for a spring. It is the minimum length at which a spring begins producing force. It is not so obvious how the zero length of a muscle could be defined. While a spring

begins to produce force when stretched beyond its zero length, a muscle only begins to actively generate force when its motoneurons become active. A logical link between motoneuron activity and muscle length is through the static stretch reflex. Consider the excitability of the motoneuron pool in a subthreshold state. If the muscle is stretched to a new length, the excitability will increase due to afferent input from muscle stretch receptors. The more the muscle is stretched, the greater the afferent input from stretch receptors. By stretching the muscle sufficiently, the excitability of the motoneuron pool will reach threshold and active generation of muscle force will begin. The muscle length at the activation threshold is its zero length. The zero length of the muscle can be voluntarily controlled by controlling the level of excitability of the motoneuron pool. The less excitable the motoneuron pool, the greater the zero length.

Active muscle force depends on the muscle length and the level of voluntary excitation of the motoneuron pool. As defined above, zero length is a measure of voluntary excitation. Therefore, a muscle behaves like a spring in that its active force will be a function of stretched length and zero length. Unlike a spring though, this relationship is not linear. Asatryan and Fel'dman (1965) measured the active muscle torque as a function of joint angle for different levels of voluntary excitation, i.e. different zero lengths. They concluded that each zero length

determined a unique torque-angle curve and that no curve
intersected any other. They called these torque-angle curves
invariant characteristics (Figure 1A). Each invariant
characteristic intersects the torque-axis at a unique angle
which is the static threshold angle for muscle activation,
i.e. the joint angle which corresponds to the zero length of
the muscle.

Fel'dman's equilibrium point theory attempts to explain
the process governing the final maintained state of a
movement. He proposes that the final joint angle or
equilibrium point is achieved by selecting a zero length
whose corresponding invariant characteristic passes through
the point in torque-angle space defined by the desired joint
angle and the active muscle torque required for equilibrium.

When two antagonistic muscles act across a joint, the
resultant invariant characteristic will be the sum of the
invariant characteristics of each muscle. The zero length
may be set independently for each muscle, allowing various
degrees of coactivation for any desired joint angle.
Movement can be effected by a shift in the resultant
invariant characteristic. The final joint angle will be the
equilibrium point on the resultant invariant characteristic.

The equilibrium point theory, as presented above, does
not describe the kinematics of a movement between two joint
angles. That would require a description of the
transformation from the initial to the final invariant
characteristic. However, establishment of the invariant
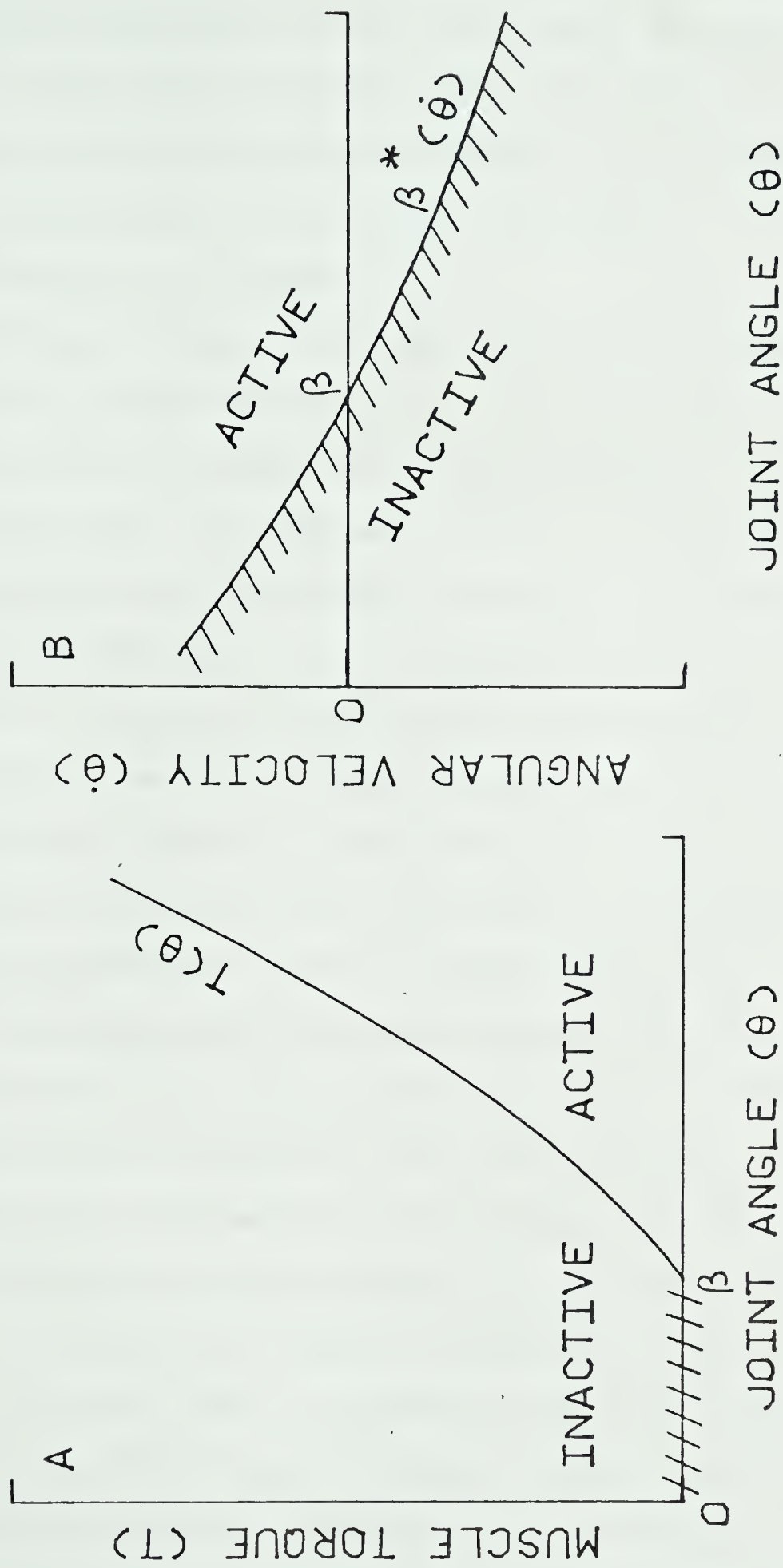
FIGURE 1. A) Invariant characteristic T(θ) with static threshold angle *B*. Muscle is inactive when θ<*B* and active when θ>*B*. Muscle length increases with θ. B) Dynamic threshold curve B*(θ̇) with static threshold angle *B*. Muscle is inactive when θ<*B** and active when θ>*B**. Muscle length varies with θ, shortening when θ<0 and lengthening when θ>0. Dynamic threshold angle *B** decreases as rate of stretch increases and increases as rate of shortening increases (adapted from Fel'dman, 1974b).

characteristic determining the final joint angle appears
relatively independent of the intervening process (Fel'dman,
1966b), so Fel'dman concludes that "the main part of the
central program of the movement seems to be reduced to a
quick establishment of the final invariant
characteristic..." (1980b).

Fel'dman (1974a, 1974b) has extended his theory to
incorporate movement kinematics. To account for the patterns
of muscle activity seen during rapid movement from one joint
angle to another, he proposes a dynamic threshold angle for
muscle activation. The dynamic threshold angle represents
the minimum length at which a muscle is active for a given
velocity of stretch or shortening. Like the static threshold
angle which determines the boundary between muscle
activation and silence in one-dimensional joint angle space,
the dynamic threshold angle determines a threshold curve or
activation boundary in the two-dimensional space of joint
angular velocity and joint angle (Figure 1B). The threshold
curve displays the characteristic feature of a reduction in
theshold with an increase in the rate of muscle stretch,
i.e. the faster the muscle is stretched, the shorter the
length at which it first becomes active. Thus, when a muscle
is being stretched the dynamic threshold is lower than the
static threshold. When a threshold curve is crossed in one
direction an active muscle becomes silent; crossing a
threshold curve in the opposite direction causes a silent
muscle to become active. The direction in which the

threshold curve of an agonist muscle must be crossed for activation will be opposite to that of its antagonist.

One way in which movement may be initiated is by a shift in the dynamic threshold angles of both muscles such that the agonist is active while the antagonist is silent. At some point during the movement the threshold curve of the agonist muscle will be crossed and it will fall silent. Reciprocal activation of the antagonist will occur when its threshold curve is crossed. Towards the end of the movement the angular velocity will decline to zero due to the combined braking action of the antagonist muscle and the visco-elastic forces of joint, tendons and muscles. Generally, though, movement will not cease when the velocity reaches zero, but will oscillate before the final endpoint is achieved. As the angular position and angular velocity change during these oscillations, the threshold curve of the agonist muscle may be crossed again, reactivating it. Fel'dman's theory would then provide a qualitative description of the triphasic pattern of muscle activity normally characteristic of rapid movements.

The validity of Fel'dman's equilibrium point theory hinges on the existence of invariant characteristics. The sufficiency of their existence to validate the theory makes it appealing. Since any point on an invariant characteristic is a potential equilibrium point and the set of invariant characteristics encompasses the entire torque-angle space, the final equilibrium point must be on an invariant

characteristic. Therefore, if invariant characteristics
exist, the movement will have begun on one invariant
characteristic and ended on another. That is the essence of
the theory. However, attempts to reproduce some of the
results reported by Asatryan and Fel'dman (1965) have been
unsuccessful (Gottlieb and Agarwal, 1983). Asatryan and
Fel'dman claimed that the final angular position attained
was independent of the previous history of the muscle, but
published only part of their data in support. Gottlieb,
however, found that unloading the muscle in two stages or
that unloading and reloading it resulted in final joint
angles which differed considerably from those obtained by a
single loading or unloading to the same final torque.
Fel'dman (1974a) appears to be aware of this hysteresis, but
fails to elaborate on its significance for his theory.

While there may be shortcomings in Fel'dman's attempt
to give his theory a physiological basis, the idea that
central nervous system control of muscles during limb
positioning is analogous to controlling the length-tension
characteristics or stiffnesses of springs, has proven
fruitful for other investigators (Polit and Bizzi, 1979;
Cooke, 1980). Cooke attempted to combine the processes
governing movement kinematics and final joint angle. He
suggested that movement is accomplished by a step change in
the net stiffness of the combined agonist and antagonist
muscles acting about a joint, i.e. the net joint angular
stiffness.

Cooke's model appears to be too simplistic. Its application is limited since it does not make a distinction between the net joint angular stiffness and the individual contributions from agonist and antagonist muscles acting about a joint. Thus, it predicts that the same final equilibrium angle will be attained as long as the sum of the individual stiffnesses remains constant, even though it is intuitively obvious that changing the individual stiffnesses while keeping the sum constant will cause the equilibrium angle to shift in the direction of action of the muscle group whose stiffness has increased. Cooke (1980) claimed that his model could provide an explanation for the linear relationship between movement amplitude and velocity which he and others (Bouisset and Lestienne, 1974; Wadman et al., 1979; Ghez, 1979; Georgopoulos et al., 1983b) have observed during rapid movement. If the initial spring stiffness was kept constant while the final spring stiffness increased progressively with movement amplitude, Cooke's model predicts this linear relationship. However, when he actually measured the net joint angular stiffness during these movements, Cooke (1982) found that the stiffness remained constant as movement amplitude increased. This result can be explained only if the ratio of the stiffness of agonist to antagonist muscle groups acting about the joint varied, while the sum of their stiffnesses remained constant.

Polit and Bizzi (1979) proposed an equilibrium point theory in which the length-tension characteristics of

agonist and antagonist muscles were individually regulated during movement in order to achieve a desired final endpoint. They found that monkeys trained to point to a target, were still capable of executing coarse positioning movements to the target location after being deprived of afferent feedback. This suggested to them that the monkeys had learned a motor program which could select, for the agonist and antagonist muscles, a set of length-tension characteristics or stiffnesses, whose equilibrium point corresponded to the target location. Although Polit and Bizzi have demonstrated, as a consequence of this experiment, that stretch reflex mechanisms are not necessary for the production of positioning movements, there is little doubt that they normally contribute to muscle stiffness (Nichols and Houk, 1976).

Fel'dman (1974b) has considered the effects of deafferenting a muscle on movement control. Loss of the static and dynamic stretch reflexes will result in a reduction of muscle stiffness which could reduce the endpoint stability of a movement, as well as its peak velocity and acceleration. Deafferentation also removes one of the possible sources for shifts in the invariant characteristic or zero length, namely, the gamma loop. Therefore, after deafferentation the range of possible equilibrium point shifts may be restricted, consequently limiting the operating range of the muscle in torque-angle space.

Polit and Bizzi (1979) postulated that the process controlling movement velocity might be independent of that controlling final joint angle. This is in contrast to Cooke's model in which both the kinematics and the final equilibrium point were determined by a single step change in net joint angular stiffness. The results of further investigations supported the view that the processes controlling position and trajectory were independent. Lestienne et al. (1981) found that final EMG activity in agonist and antagonist muscles correlated well with the final position, but not the velocity, direction or amplitude of the movement which correlate well with the initial EMG activity, particularly in rapid movements. Their results also suggested that final joint angle could be coded in the central nervous system as the ratio of activity in antagonistic muscles acting about a joint.

Both Fel'dman and Cooke assume that the shift to the final equilibrium point or final muscle stiffness occurs in a step-like manner. Bizzi et al. (1982) tested this hypothesis for movements performed by monkeys. They found that the equilibrium point shifted gradually rather than abruptly, but their observations were restricted to movements made at moderate speeds. As they pointed out, rapid movements are generated by a more step-like shift in the equilibrium point.

The equilibrium point hypothesis, as proposed by Polit and Bizzi, has been criticized by Day and Marsden (1982) and

Rothwell et al. (1982). They reasoned that if Polit and Bizzi were correct in postulating a centrally programmed selection of agonist/antagonist muscle stiffnesses then the unexpected alteration of a viscous load, although affecting the movement kinematics, should not affect the final equilibrium position. Day and Marsden tested this prediction using a thumb flexion paradigm in which subjects' position sense had been impaired by anesthesia of joint and cutaneous afferents. When a viscous load was unexpectedly changed, subjects produced consistent errors in final angular position. Similar results were obtained with a pathologically deafferented subject in the study by Rothwell et al. (1982). In addition, the deafferented subject showed a complete inability to compensate for an unexpected external disturbance which halted movement at a point halfway to the target for a period of 200 milliseconds. This is contrary to the findings of Polit and Bizzi (1979) and Bizzi et al. (1982) that perturbations had little effect on a deafferented monkey's ability to successfully achieve a designated angular position of the elbow, regardless of the point in the movement trajectory at which the disturbance occurred. One possible explanation for this discrepancy is that movement of the distal phalanx of the thumb may be subject to a large amount of nonlinear Coulomb friction, relative to its inertia. The friction arises from long tendons which couple the thumb to muscles in the forearm. Movement of the forearm is achieved through comparatively

shorter tendon linkages, while the moment of inertia is at least two orders of magnitude greater than that of the distal phalanx of the thumb. Consequently, once slowed down or stopped, the distal phalanx of the thumb would be less likely to continue moving to the intended position than the forearm unless additional muscle force was developed to overcome the 'stickiness' of the Coulomb friction.

Impulse-Variability Theory

The equilibrium point theory and the impulse-variability theory are not mutually exclusive; rather, they tend to focus on positioning movements from different viewpoints. While the former directs attention primarily to observed movement trajectories and maintained equilibrium endpoints, the latter is more concerned with the processes underlying variability in endpoints, which impose limitations on accuracy.

Observations that provided much of the groundwork from which the impulse-variability theory has arisen were made by Woodworth (1899) and Fitts (1954). Woodworth found that aimed movements were composed of two distinct segments: an initial-impulse phase and a current-control phase. He regarded the initial-impulse phase as preprogrammed and ballistic, while the current-control phase relied on visual feedback to correct for any deviations from the intended path. Both phases contributed to a tradeoff between speed and accuracy. Fitts formalized this tradeoff quantitatively

in a form which has become known as Fitts' Law.

Schmidt et al. (1979) proposed a model to explain the speed-accuracy tradeoff in relation to the initial-impulse phase of a rapid movement. They examined movements which were restricted to an initial-impulse phase with no time allowed for additional corrections, i.e. no current-control phase. They found that the error in final position varied linearly with the speed of subjects' movements. The standard deviation of the final position error (which they called the effective target width) varied directly as the distance to the target and inversely as the average time taken to make the movement. In other words, the effective target width was directly proportional to the average velocity of the movement. To explain the observed linear relationship between speed and error, Schmidt et al. proposed an impulse-variability theory. According to this theory, rapid, target-directed movements are produced by the generation of a force impulse in the agonist muscles which propels the limb toward the target. The force impulse can be represented by a force-time curve with a characteristic shape that can be scaled along either the force or time axis. Essentially, the theory assumes that subjects adapt the force impulse to the requirements of a task by appropriately scaling it in force and time. The scaling parameters for force and time are assumed to be random variables whose standard deviations are proportional to their means. From this, it follows that the variability in the force and time parameters will

determine the effective target width, i.e. the variability in final position.

Although the data presented by Schmidt et al. supported the empirical aspects of their theory, Meyer et al. (1982) pointed out several serious flaws in its derivation. The errors resulted from oversimplification of movement dynamics, misapplication of physical laws and violations of probability theory. The corrected version of the theory actually predicted that the effective target width would be independent of movement time, i.e. it would be a linear function of target distance only. Since this was not in accordance with observations, Meyer et al. went on to develop a new impulse-variability theory which avoided the shortcomings of the previous one and could account for the observed linear speed-accuracy tradeoff. Like the theory of Schmidt et al., it assumes that an aimed movement is produced by generating a force impulse whose magnitude and duration are dependent on the values of a force parameter and a time parameter. It assumes that a prototype curve exists which can be scaled in force and time without affecting its shape, i.e. the scaling process is linear (Figure 2). Whereas Schmidt et al. were concerned mainly with the acceleration phase of the force impulse, Meyer et al. put equal emphasis on the deceleration phase. They proposed that the impulse curves are symmetric with equally strong acceleration and deceleration phases. The second half of each curve is an inverted mirror-image of the first. In
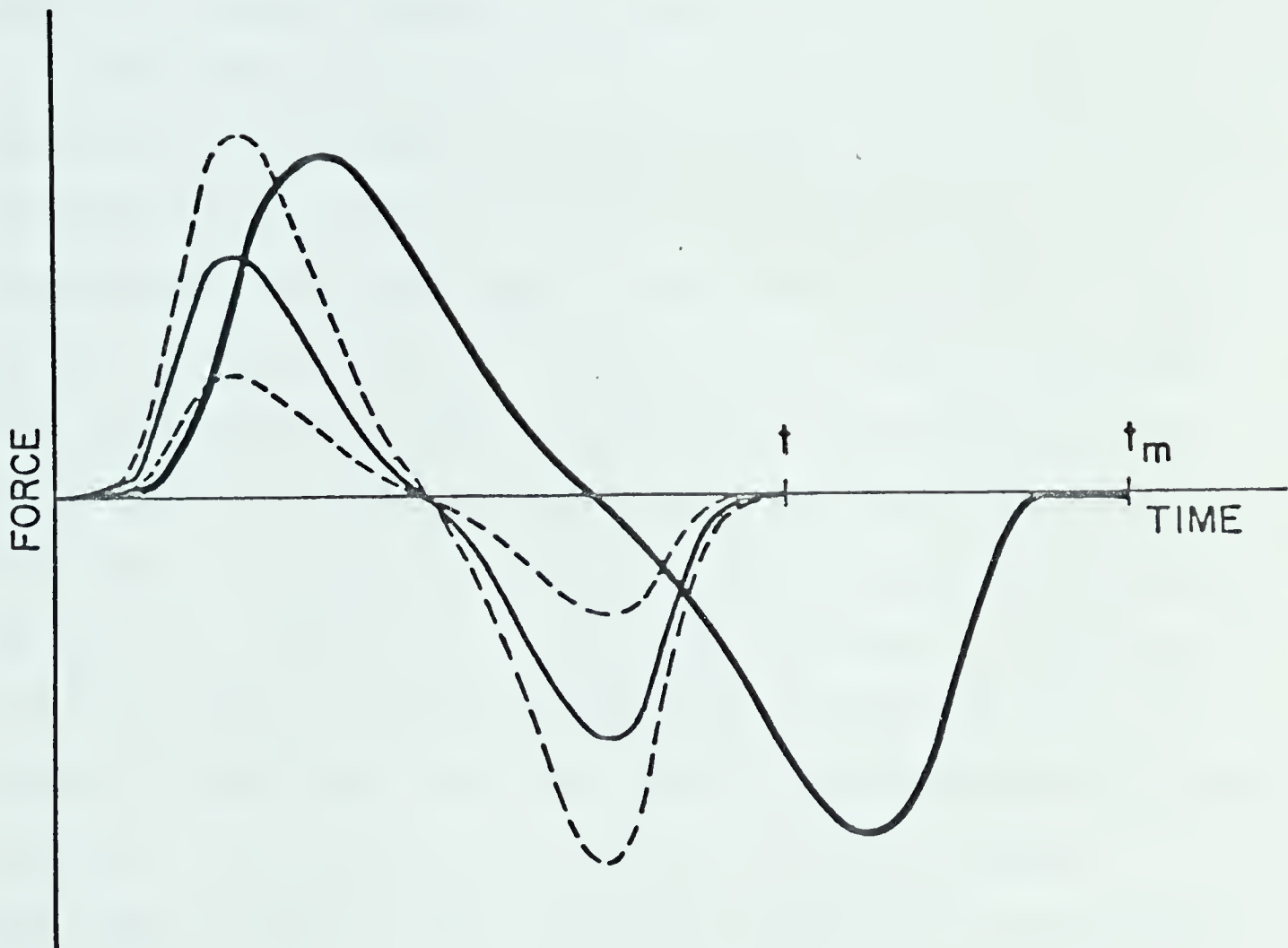
FIGURE 2. The thick solid curve to the right represents a prototype time function that can be time scaled or force scaled. The thin solid curve to the left represents a second time function obtained from the first by time scaling. Note that time scaling involves both a compression along the time axis and a reduction in amplitude. The two dashed curves represent force-time curves obtained by force scaling the second time function (Meyer et al., 1982).

order to account for the linear speed-accuracy tradeoff, the new theory specifies the precise shape of the force impulse curves, i.e. the shape is determined as a unique function of time. It, therefore, predicts the precise shape of theoretical movement trajectories, something which was not possible from the theory formulated by Schmidt et al.

The symmetric impulse-variability theory proposed by Meyer et al. is given substantial support from experimental observations. Bouisset and Lestienne (1974) have presented evidence for both the symmetry and uniqueness of the shape of the force-time curves. Flament et al. (1982) suggested that there might be some variability in the symmetry from trial to trial, but the movements which they studied may not have been of a ballistic nature. The postulated scalability of the force-time curves by a force parameter is supported by the studies of Freund and Büdingen (1978) and Ghez and Vicario (1978a) who have shown that in tasks requiring rapid isometric force adjustments, there is often a simple amplitude scaling of the force-time curves which preserves their basic shape. In addition, Mishima et al. (1981) have shown that when movements are made as fast as possible, switching occurs from scaling of acceleration amplitude only (force scaling) to scaling of acceleration both in amplitude and time, as movement amplitude increases. Finally, the agreement between theoretically predicted movement trajectories and those observed experimentally is remarkably good.

Meyer et al. (1982) have not attempted to determine how their force-time curves might emerge from known biomechanical and neurophysiological mechanisms. However, they draw attention to the fact that their theory can be made compatible with the equilibrium point theory. Schmidt and McGowan (1980) had argued that the failure of unexpected load perturbations to cause systematic errors in final position, while dramatically altering movement times, was contrary to predictions of the impulse-variability theory, but supported the equilibrium point theory. Meyer et al. countered the arguments of Schmidt and McGowan by accusing Schmidt (1980) of placing an unjustified constraint on the impulse-variability theory, namely, that the force impulse must have an invariant time course regardless of movement perturbations. They went on to demonstrate that, in principle, their force-time curves could be achieved by appropriate adjustments of stiffnesses and/or resting lengths of the agonist and antagonist muscles. The muscles would then respond to an unexpected external perturbation by generating a net internal force which would alter the time course of the force impulse. Meyer et al. concurred with Lestienne et al. (1981) in concluding that rapid movements include two independent process: a phasic process or initial impulse which quickly adjusts the stiffnesses of the muscles at the movement onset and a tonic process responsible for maintaining the limb at the final equilibrium position. Their symmetric impulse-variability theory characterizes the

kinematics of the initial impulse in the absence of perturbations.

Wallace (1981) has focused on the implications of an impulse-timing theory for timing and intensity of activity in the agonist and antagonist muscles responsible for generating the force impulses. He refers to the theory as an impulse-timing theory, while acknowledging that there can be independent regulation of both the amplitude and duration of the impulse. In fact, the theory which he discusses is an impulse-variability theory. According to him, the theory can be summarized as follows: 1) the average velocity of movement will determine the sizes of the acceleration and deceleration impulses; 2) movement time as opposed to movement distance will determine the duration of the acceleration impulse; 3) this duration will always be 50% of the total movement time. From this he deduces four postulates about the timing and intensity of activity in agonist and antagonist muscles.

The first postulate states that "the duration of the initial agonist burst and onset of antagonist activity will be positively related to the total movement time." A subsequent study by Wallace and Wright (1982) supported this hypothesis, but when viewed in light of other investigations it appears to be only part of the total picture. Seen as a whole, they suggest that there is a degree of flexibility in the relationship between timing of muscle activity and movement time. Lestienne (1979) showed that for movements

which were fast enough to be considered ballistic, the agonist burst duration remained relatively constant over a two- to threefold range of movement times and amplitudes. The antagonist onset time showed a slight increase with increasing movement time which was independent of movement amplitude. Wadman et al. (1979) and Mishima et al. (1981) looked at movements made as fast as possible. They found that small amplitude movements (smaller than those examined by Lestienne) which had relatively constant movement times also had constant antagonist onset times, whereas movements of larger amplitude with progressively longer movement times had similar increases in antagonist onset times. Brown and Cooke (1981) observed the same constancy of agonist burst duration as Lestienne, but over a greater range of movement amplitudes. However, they saw a sharper rise in antagonist onset time with movement time. Marsden et al. (1983), whose data were more comprehensive than those of Brown and Cooke, showed that antagonist onset time becomes more constant as movement amplitude increases, in agreement with Lestienne's observations which were made on relatively large amplitude movements. It appears, therefore, that in rapid movements, the duration of the agonist burst remains relatively constant while adjustments are made in the intensity of the burst to produce different initial accelerations. This conclusion is also supported by the investigations of Freund and Büdingen (1978), Hallett and Marsden (1979), Hallett and Khoshbin (1980) and Hoffman and Strick (1982). As movement

amplitude becomes large antagonist braking tends to occur progressively later, resulting in longer movement times. For small movement amplitudes, the antagonist onset time is more closely related to movement time. Thus, the first postulate is partly, but not totally correct.

The second postulate states that "the ratio of the duration of the initial agonist burst to the total movement time will be unaffected by changes in movement length, movement time or inertial load of the movement." Neither the data of Wallace and Wright (1982) nor those of the investigators cited above supports this hypothesis. First, the agonist burst duration is constant over a wide range of movement times. Hence, the ratio of agonist burst duration to movement time varies inversely with movement time. Secondly, since antagonist onset time becomes more constant as movement amplitude becomes large, while movement time concurrently increases, the ratio of antagonist onset to movement time will vary both with movement time and amplitude. The second postulate is, therefore, invalid.

The third postulate states that "the intensity of the initial agonist and antagonist burst will be positively related to the velocity of the movement." The first part of this postulate enjoys considerable support. There seems to be a consensus that both the amplitude and the integrated area of the initial agonist burst is well-correlated with peak velocity under almost all movement conditions to which the theory applies (Bouisset and Lestienne, 1974; Lestienne,

1979; Hallett and Marsden, 1979; Brown and Cooke, 1981; Hoffman and Strick, 1982; Marsden et al., 1983). There is at least one circumstance though, where this is not true. Mishima et al. (1981) showed that for large amplitude movements made as fast as possible, a point is reached where the amplitude of the agonist burst saturates and further increases in velocity can occur only by delaying the onset of the antagonist. However, this is a limiting case and does not undermine the general validity of the hypothesis.

The second part of the postulate, related to the intensity of the antagonist burst, must be qualified. Hoffman and Strick (1982) and Marsden et al. (1983) have shown that when small amplitude movements, having short movement times, are made with the same peak velocity as larger amplitude movements, having longer movement times, the small amplitude movements have larger antagonist bursts. A positive relation between antagonist burst intensity and movement velocity holds only when movements are of the same amplitude and begin from the same joint angle. When single-joint movements of similar amplitude and velocity are produced along different arcs less antagonist activity will be required to halt those movements which end nearer the extremes of joint rotation (Marsden et al., 1983) because extra braking force is supplied by the active length-tension properties of the stretched antagonist muscles, as well as from the visco-elastic properties of the joint, tendons and muscles which increase in a nonlinear fashion near the

extremes of joint rotation.

The final postulate states that "the intensity of the initial agonist burst will be positively related to changes in inertial load when movement velocity is held constant." This statement is almost a truism. It follows immediately from the first part of the third postulate. Changing inertial load for a given velocity requires a similar change in the accelerative force impulse as a change in velocity for a given load. The results of Bouisset and Lestienne (1974) and Lestienne (1979) confirm this.

The fact that some of Wallace's postulates are weak or seemingly invalid does not necessarily cast doubts on the symmetric impulse-variability theory of Meyer et al. (1982). It does point out some flaws in his reasoning though. Basically, he makes too strong a link between movement time and duration of the agonist burst. This is because he fails to take into consideration the fact that the braking process is influenced both by the time of onset of the antagonist burst and its intensity. Clearly, the agonist burst is regulated in a pulse-height rather than a pulse-width fashion. Taken together, the data of Hallett and Marsden (1979) and Marsden et al. (1983) suggest that the same holds true for the antagonist burst. Wallace correctly deduces that the timing of antagonist onset is important, but he relates this to movement time only, when, in fact, it is related both to movement time and amplitude. Marsden et al. (1983) show that to achieve a faster velocity (shorter

movement time) while moving through the same amplitude, not only must the pulse-height of both the agonist and antagonist increase, but the antagonist onset must be earlier. As movement amplitude becomes progressively larger, the strategy changes because muscle activity saturates and the visco-elasticity of the joint, tendons and muscles begins to play a more prominent role in braking than for smaller amplitude movements. This reinforces the suggestion by Bizzi et al. (1982), that the central nervous system takes into account both length-tension and biomechanical properties of muscles and joints in deriving control strategies for movement.

C. Brain Motor Centers

Although the the execution of motor programs governing rapid movements has been well-characterized peripherally, the nature of the central representation of these programs remains obscure. A number of brain areas have been implicated in the initiation and execution of rapid movements, but little is known about the transformations between neuronal activity and motor output. General ideas about the function of various brain centers involved in motor control have, nevertheless, emerged as the result of observations of motor deficits suffered in pathological conditions or following surgically induced lesions, as well as from direct recordings of neuronal activity.

Allen and Tsukahara (1974) proposed a scheme regarding the roles of various brain structures in movement (Figure 3). It is basically a three-stage model of motor control. The lowest stage is the executive level involving the motor cortex and the intermediate cerebellum which is thought to update motor commands in a continuous fashion, on the basis of proprioceptive feedback from the moving limb. The second level is concerned with the planning and programming of the motor commands. This is thought to occur through two main pathways linking the cortical association areas with the motor cortex. One involves the cerebellum, the other the basal ganglia. In addition, association areas can influence the motor cortex directly. At the highest level are the events which precede planning and are presumably manifestations of the intention to move. Allen and Tsukahara have not attempted to locate this final stage in any particular brain structure. Paillard (1982) regards it as being a 'mysterious' process.

Basal Ganglia

Much of what has been proposed regarding the role of the basal ganglia in motor control is based on observations of the motor disorders of Parkinson's disease. Kornhuber (1974) proposed a model for motor control in which the basal ganglia functioned as a ramp generator, i.e. as a generator of smooth movements which could be performed at any chosen speed, in particular, slowly. The cerebellum, on the other
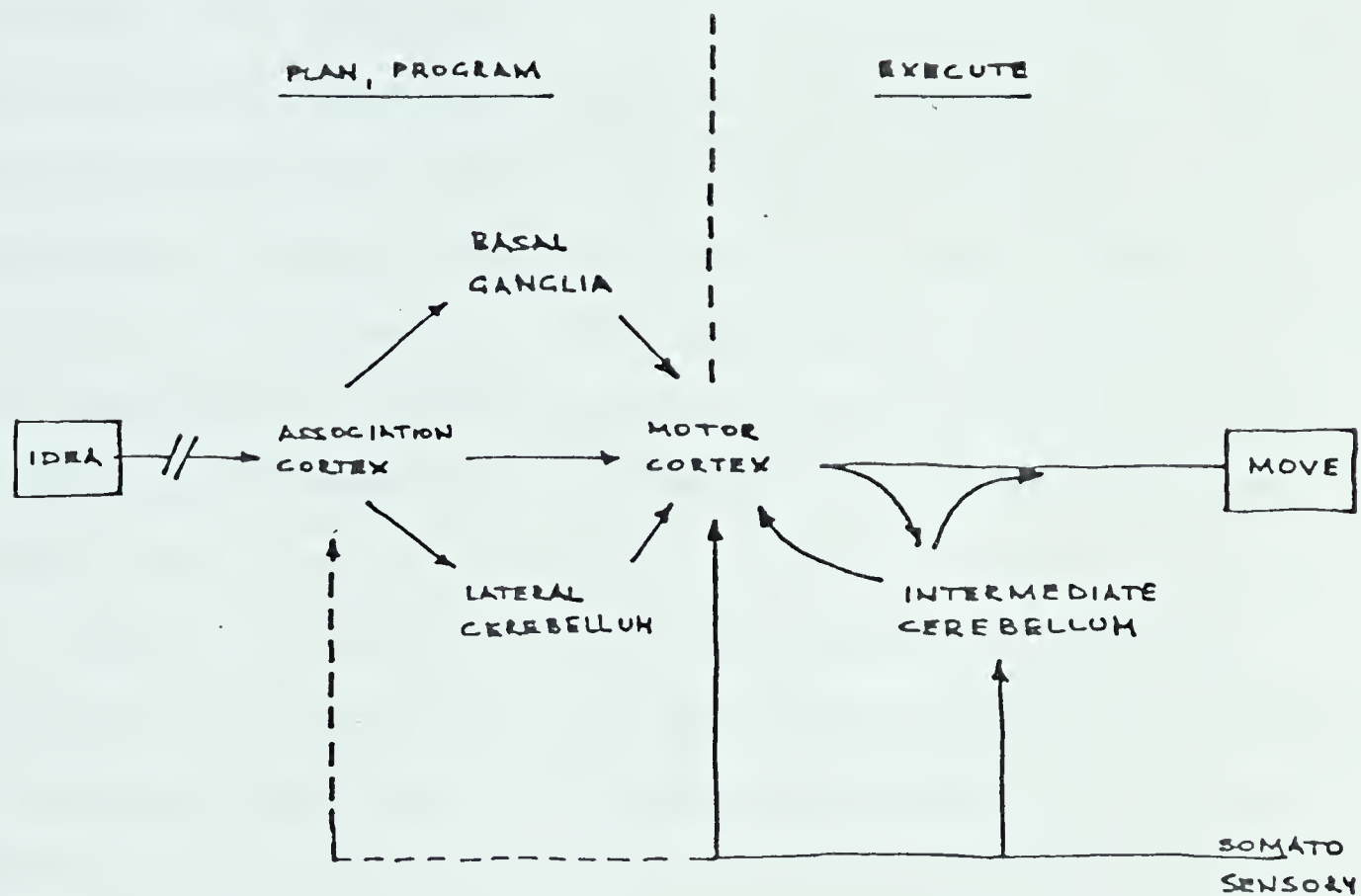
FIGURE 3. Scheme showing the proposed functions of several brain structures in motor control (adapted from Allen and Tsukahara, 1974).

hand, was responsible for generating rapid, ballistic movements. The evidence which he presented as positive support for viewing the basal ganglia as a ramp generator was twofold. First, he perceived much of the akinesia seen in Parkinson's disease as arising in situations where initiation or change of ramp movements was essential. Secondly, he cited the work of DeLong and Strick (1974) who had seemingly shown that neurons in the putamen and globus pallidus fired more often in relation to ramp than ballistic movements. Although there has been no direct evidence to contradict the view that the basal ganglia participate in the generation of ramp movements, there is evidence which challenges Kornhuber's dichotomy of function between the basal ganglia and cerebellum.

DeLong and Georgopoulos (1981) point out that many neurons in the basal ganglia discharge in relation to rapid movements, therefore, precluding an exclusive role for the basal ganglia in the generation of slower ramp movements. They also draw attention to the fact that simply because neuronal discharge covaries with slow movements, for example, does not imply that this activity is related to processes controlling movement speed. It may instead reflect involvement in a process such as afferent feedback control.

Hallett and Khoshbin (1980) demonstrated, moreover, that ballistic movements were often abnormal in Parkinson's disease. While the reciprocal pattern of agonist/antagonist activation appeared temporally normal, patients with

Parkinson's disease showed signs of a limited ability to increase agonist burst amplitude. As a result, their movements were often slow and were sometimes characterized by a series of alternating bursts subsequent to the three bursts of the normal triphasic pattern. Prolonged bursting tended to occur more often for larger amplitude movements. They went on to suggest that there might be two separate processes involved in the production of a movement. One, which they called 'timing' was concerned with timing, sequencing and relative amplitude of muscle activation. They attributed this role, at least in part, to the cerebellum. The other process, which was termed 'energizing' was responsible for selecting the appropriate muscles for a particular movement and setting the absolute intensity of muscle activation. It should be noted that 'energizing' included the inhibition of inappropriate muscles. In their view, 'energizing' was the function of the basal ganglia. Finally, they argue that virtually every type of movement dysfunction resulting from disorders of the basal ganglia can be explained as a consequence of a derangement in the 'energizing' process.

Some more direct evidence for 'energizing' comes from work by Horak and Anderson (1982) who showed that both kainic acid-produced lesions and microstimulation in the golbus pallidus prolonged the duration of rapid reaching movements made by monkeys, but did not delay movement initiation. The magnitude and buildup of EMG activity in the

different muscles participating in the movement was simply scaled without affecting the sequential organization of muscle activation.

Observations made by Hore and Vilis (1980) on movement disorders induced by cooling the golbus pallidus are also compatible with the concept of 'energizing.' A common cause for the observed disorders appeared to be an early onset of antagonist muscle activity, at a higher level of intensity and with a longer duration, than normal. This prolonged activity was usually accompanied by coactivation of the agonist muscles which suggested that the absolute intensity of antagonist activation was set too high and that this had caused an earlier and more powerful recruitment of antagonist motoneurons than normal. The subsequent failure to achieve the correct balance of agonist/antagonist activity further impaired the movement.

Georgopoulos et al. (1983b) have observed movement-related activity in the globus pallidus and subthalamic nucleus of the basal ganglia. They suggested that this activity was derived from two processes. The primary process was a step change in the rate of firing which was related to movement direction. In addition, there was often a secondary process producing modulation which varied with movement amplitude. They suggested that this activity might have a facilitatory action on populations of neurons in the motor cortex since: 1) it occurs later in the basal ganglia than corresponding movement-related activity

in the motor cortex; 2) there are anatomical pathways which would allow afferent input from the motor cortex to be processed and sent back to precentral motor areas; 3) microstimulation of the globus pallidus slows the ensuing movement when delivered just prior to movement onset, i.e. just after the first changes in e.m.g. (Anderson and Horak, 1981); 4) lesions and cooling of the globus pallidus prolongs movements without affecting reaction times (Hore and Vilis, 1980; Horak and Anderson, 1980). This is in agreement with the view of Denny-Brown and Yanagisawa (1976) that the basal ganglia are in a position to continuously sample activity projected from cortical areas and can facilitate one particular action while suppressing others.

Marsden (1982) has conceived the basal ganglia as being primarily involved in motor planning. His motor plan is a type of operating system which manages the execution of motor programs. On the basis of an objective and a perceptual judgment of the state of the environment (both internal and external), it assembles and sequences motor programs to produce the desired output. The basal ganglia draw upon a library of small motor programs stored elsewhere, deciding at what point in space-time each one should be executed. The motor plan is able to monitor the resulting movements in order to make adjustments, if necessary. Marsden's view is based largely on clinical observations of patients with Parkinson's disease. He does not see them as having deficits in perceptual judgment or in

the ability to execute simple movement sequences, i.e. motor programs, but they appear unable to effectively organize the concurrent or sequential execution of several motor programs. Thus, they are impaired in their ability to execute simultaneous movements, change criteria in motor tasks with alternate possibilities, correct inappropriate responses and use predictive control during visuomotor tracking. Marsden has taken a more global view of Parkinson's disease than Hallett and Khoshbin (1980), who concluded from their observations that the basal ganglia were primarily involved in the process of 'energizing.' In Marsden's scheme, 'energizing' could be considered a secondary function, i.e. a motor program that is both resident in the basal ganglia and managed by the basal ganglia.

Cerebellum

As noted above, the role of the cerebellum in motor control appears to be distinct from that of the basal ganglia. It is also more clearly defined. Brooks and Thach (1981) have revised the original scheme of motor control proposed by Allen and Tsukahara (1974), as it pertains to the cerebellum. In their revision, the cerebellum functions in concert with the primary motor cortex to prepare and execute simple movements by triggering small, stored programs which are primarily concerned with the timing of agonist and antagonist muscle activity. They point out that

such control is advantageous when feedback corrections are unnecessary. It permits execution to occur faster and in a more continuous fashion than would be possible using feedback regulation. They also suggest that the cerebellum may serve as a comparator of central and peripheral information for the early detection and correction of motor errors.

Most of the movements which Brooks and Thach consider in relation to cerebellar control are 'simple' movements about a single joint. They have further classified these 'simple' movements according to the manner in which they are arrested. The term 'ballistic' is used to refer exclusively to very rapid movements which are terminated without the action of antagonist muscles. Such movements are arrested either by a mechanical stop or by the combined visco-elastic retarding forces of the joint, tendons and muscles. As previously noted, these movements can be executed by means of a single agonist burst. 'Simple' movements which involve antagonist muscle activation are termed 'self-terminated' movements.

Brooks and his colleagues have made extensive use of the technique of brief local cooling to induce reversible focal lesions in the monkey cerebellum. Conrad and Brooks (1974) looked at rhythmic, alternating 'ballistic' arm movements made between two mechanical stops. On cooling the dentate nucleus, they found that the duration of each burst of muscle activity was prolonged while the intensity

remained about the same. This resulted in more time being spent at the extremes of the movement range and hence, increased the cycle time. It appears, therefore, that the cerebellum governs the timing, but not the intensity of muscle activity in these movements.

Studies of 'self-terminating' movements in patients with cerebellar deficits (Hallett et al., 1975b; Marsden et al., 1977) have shown that movement initiation is delayed and burst duration in the agonist and/or antagonist muscles is prolonged. In addition, when the antagonist muscles were preloaded, the normal inhibition of antagonist muscles seen prior to activation of the agonists was delayed or absent in most of these patients. Terzuolo and Viviani (1974) studied movements in which the resistive load opposing a near-maximal voluntary isometric contraction suddenly gave way and decayed to almost zero. Patients with cerebellar disorders were less successful in braking such movements than normal subjects. Failure could be attributed to their inability to quickly silence agonist muscle activity and activate antagonist muscles. Agonist activity was, therefore, prolonged and antagonist onset delayed with respect to normal subjects. These findings support the idea that the cerebellum is involved in 'timing,' conceived in the sense used by Hallett and Khoshbin (1980).

A delay in movement initiation, similar to that seen in patients with cerebellar disease, is seen in monkeys following cooling of the dentate nucleus (Meyer-Lohmann et

al., 1977). Records of activity from precentral neurons suggest that this delay occurs because the cerebellum is late in issuing a timing trigger to the motor cortex. The timing trigger may originate as a movement command from the association areas, subsequently processed by the cerebellum and relayed to the motor cortex via the thalamus. In a cerebellectomized monkey this trigger was delayed, presumably because alternative pathways which were slower had to be used (Lamarre et al., 1978).

Terzuolo and Viviani (1974) have shown that during a sudden-release movement which subjects were instructed not to voluntarily arrest, a burst of activity, coincident with peak acceleration, occurred in the antagonist muscles and simultaneously the agonist muscles fell silent. The amplitude of the burst was correlated with the magnitude of the peak acceleration. Both the antagonist burst and agonist silence were abolished by dorsal rhizotomy in monkeys (Terzuolo et al., 1974), implying that this patterned activity normally resulted from segmental reflex actions. Lamarre et al. (1978) concurred, since they found that antagonist inhibition, which normally accompanies the first agonist burst in rapid, 'self-terminated' movements, disappeared following limb deafferentation. However, the absence of a silent period in the agonists and the failure of antagonist activity to be tightly coupled to acceleration in cerebellar patients (Terzuolo and Viviani, 1974), indicates that these segmental responses may normally be

regulated by the cerebellum. This is further supported by the observation that while a deafferented, cerebellectomized monkey could generate patterns of muscle activity which were qualitatively similar to those seen in a deafferented monkey with an intact cerebellum, it was unable to time the antagonist activity appropriately (Lamarre et al., 1978). Terzuolo and Viviani (1974) found that normal subjects prolonged and intensified the antagonist burst when instructed to voluntarily arrest sudden-release movements. Cerebellar patients did not do this and consequently failed in their attempts to arrest these movements. This suggests a role for the cerebellum in controlling the reciprocal behavior of agonist and antagonist muscles, as well as predictively determining optimal times for the modification of ongoing segmental activity, particularly for the purpose of braking movements.

There is evidence that the interpositus nucleus may have such a predictive capacity. It receives inputs both from the motor cortex and peripheral sensory receptors and could conceivably act as a comparator of the two signals. Furthermore, Burton and Onoda (1978) and Soechting et al. (1978) have found that the discharge of interpositus neurons receiving peripheral proprioceptive input often shows considerable phase advance with respect to angular displacement. This would allow the interpositus nucleus to provide dynamic control of an evolving movement.

Vilis and Hore (1980) have examined antagonist muscle activity which occurs when a torque pulse briefly displaces the arm of a monkey trained to maintain a target position. The agonist muscles normally respond to the perturbation with a burst of activity that causes the arm to move back toward its original position. This activity is closely followed by a burst of activity in the antagonist muscles, timed to occur slightly before or just after the beginning of antagonist stretch in order to actively brake the return movement. As the dentate and interpositus nuclei were cooled the onset of antagonist activity became delayed and its duration prolonged. This led to the production of a series of overcorrections in the form of undamped oscillations or tremor. To further investigate this degeneration in the braking process, Vilis and Hore examined the responses of precentral neurons whose activity was reciprocally related to the direction of initial stretch. They found that these units responded with a burst of activity that preceded activity in the antagonist muscles and often occurred even before the muscles were stretched. Following cooling this response was delayed until after the onset of antagonist activity.

They suggested two ways that the cerebellum might function to facilitate braking. The cerebellum could provide the motor cortex with information that is phase advanced in relation to movement. As a result, the activity in precentral neurons begins prior to muscle stretch and will

generate muscle activity which precedes stretch reflex activity. In addition, there is an inhibition of long latency stretch responses which prevents muscle activity from being prolonged and thus, acts to prevent the initiation of subsequent oscillations. During cerebellar dysfunction braking becomes an unregulated reflex process which breaks into oscillation. Alternatively, the motor cortex might respond to stretch of the agonist muscles by sending a signal to both the spinal cord and cerebellum. With practice, the cerebellum could learn to generate an appropriately timed command to the motor cortex for activation of the antagonist muscles in advance of antagonist stretch.

Further evidence that the dentate nucleus is involved in generating adaptive motor responses has been obtained by Hore and Vilis (1982). They trained monkeys to resist a torque pulse applied to the arm. Once a stereotyped response had been established, they occasionally substituted torque steps for the expected torque pulses. The procedure was later reversed after the monkeys had been trained to prepare for torque steps, i.e. pulses were then occasionally substituted for the expected steps. The responses to unexpected steps or pulses were different from those observed for expected steps or pulses. The former were interpreted as being more purely reflexive than the latter. When the dentate nucleus was cooled, the responses to expected pulses or steps changed. Qualitatively, they were

more like the responses to unexpected pulses or steps seen previously. These results indicate that the dentate nucleus is required for the expression of learned adaptive responses. The failure of cerebellar patients to show adaptation of reflex responses to changing conditions of postural stability (Nashner and Grimm, 1978), further reinforces the idea that the cerebellum plays a role in adaptive motor learning.

Motor Cortex

The motor cortex is the chief executive locus for motor control. While the basal ganglia and cerebellum seem to be involved in sequencing and structuring motor commands, the motor cortex functions more like a processor, switching, gating and integrating information according to instructions originating elsewhere. However, it is considerably more than a simple switchboard which directs commands to motoneurons. The motor cortex receives inputs from secondary motor areas of the cortex, including association, sensorimotor, supplementary motor and premotor areas, as well as from the cerebellum, basal ganglia and spinal cord, via the thalamus. Its outputs are able to influence many of its inputs by way of collaterals from pyramidal tract fibers directed to the thalamus, the dorsal column nuclei and spinal interneurons. Such recurrent loops certainly suggest that the motor cortex operates in an internal feedback control mode. There may be other reasons for pyramidal actions on ascending sensory

signals though, particularly since this information is not directed exclusively to the motor cortex. It may be desirable to filter or sharpen sensory data in order to focus motor commands to a part of the body whose movement is being precisely controlled. Internal feedback might also be used to provide a 'sensory image' of a motor command which might be important in error correction or detection. Wiesendanger (1981) and McCloskey (1981) discuss some of these 'sensory' functions of the motor cortex.

Returning to the executive function of the motor cortex, considerable attention has been focused on the relationships between firing patterns of cortical neurons and parameters of motor responses. Close correlation is to be expected because motor cortical cells project more or less directly to motoneurons. Discharge rates of particular cells have been shown to be correlated with velocity, acceleration, force and/or rate of change of force (Evarts, 1968; Humphrey et al., 1970; Schmidt et al., 1975; Smith et al., 1975; Lamarre et al., 1978; Cheney and Fetz, 1980; Hamada, 1981; Evarts et al., 1983). Lamarre et al. (1978) were able to show a similar correlation of discharge rate to velocity and acceleration in both intact and deafferented monkeys, providing convincing evidence that their observations were not simply due to peripheral sensory feedback modulation of the motor cortical cells. The response of a particular cell to movement or change in isometric force is generally quite stereotyped. However, a

variety of cell types, classified according to their streotyped discharge patterns, have been found. Some cells responded to both the dynamic and static phases of muscle activation; others responded to only one phase or the other (Smith et al., 1975; Conrad et al., 1977; Hepp-Reymond et al., 1978; Cheney and Fetz, 1980). A common observation has been that cells which fired tonically during the static (holding) phase, often showed a monotonic relation between discharge rate and static torque.

There is a strong implication that a tight coupling exists between the discharge rate of motor cortical cells and the intensity of muscle activation. Caution must be exercised though, in coming to such a conclusion since phasic activity in motor cortical cells often more closely resembled the rate of change of the integrated activity of target muscles than the integrated activity itself. Furthermore, there were cells which showed ramp increases in activity while muscle activity remained relatively constant (Cheney and Fetz, 1980). It is possible, nevertheless, to retain the view that motor cortical cell discharge rate reflects muscle activation. The phasic activity may be linked to motoneurons that fire phasically when muscle force changes, while the ramp activity may counteract the effects of adaptation of firing rate in tonically active motoneurons. It is more difficult to explain the activity in cells which fired much more weakly during ballistic movements, than during ramp-and-hold movements made at lower

peak velocities, requiring less activity in target muscles.
It may be due to a difference in the central control
strategy for the two types of movement and may partly be
explained by the observation that peripheral input to some
motor cortical cells is less influential during ballistic
than during controlled movements (Evarts and Fromm, 1977).
Fetz et al. (1980) issued the following warning about the
danger of being too narrow-minded in interpreting the
activity of single neurons.

> The search for neural correlates of behavioral
> functions often assumes that such functions are
> recognizably coded in neural response patterns.
> Since behavior is the consequence of interaction
> between widely distributed neurons, their individual
> responses need not resemble particular behavioral
> parameters any more than the spatial patterns on a
> holographic plate resemble the image produced by its
> proper illumination.

With this in mind, it should be noted that motoneurons
are accessible to the motor cortex via pathways other than
those that activate them directly. One of these pathways
involves the red nucleus and the rubrospinal tract. Recent
studies (Ghez and Vicario, 1978b; Kohlerman et al., 1982)
have shown that neurons in the red nucleus, like the motor
cortex, modulate their activity in advance of movement onset
in relation to parameters of movement. Since both pyramidal
and rubrospinal fibers project to spinal interneurons,
either directly or by way of collateral fibers, one of the
functions of the motor cortex may be to use neurons of the
propriospinal apparatus and segmental reflexes in activating
motoneurons. In this way, coordinated spinal activity at

multiple levels may be employed in the production of synergistic muscle actions. Propriospinal circuits, in themselves or in combination with multisegmental branching of descending tracts, may represent the anatomical substrate of movement subroutines that can be executed in various combinations, depending on the spatial and temporal patterns of descending activity. The directionally sensitive motor cortical cells observed by Georgopoulos et al. (1982, 1983a) may command such actions. While it is possible that they produce muscle synergies by direct actions on several motor nuclei, it may well be that these neurons invoke synergistic muscle actions through existing propriospinal circuits when generating movements along certain spatial trajectories. Such a mode of operation is available to the cerebellum, as well, since cerebellar outputs have access to the rubrospinal, vestibulospinal and reticulospinal tracts. This would be a potential means whereby the cerebellum could regulate adaptive postural responses of the type that are impaired as the result of cerebellar dysfunction (Nashner and Grimm, 1978).

D. Kinesthesia

The sensation of joint movement or kinesthesia is mediated through the actions of peripheral sensory receptors stimulated as the result of the movement. This is apparent from the observations that kinesthesia is impaired in the fingers when their digital nerves are blocked (Goodwin et

al., 1972) and can be abolished if the muscles are disengaged as well (Gandevia and McCloskey, 1976). This afferent information must be accessible to sensory areas of the brain in order for there to be a conscious awareness of movement. Transection of the posterior columns, anterior and lateral spinothalamic tracts and ventral and dorsal spinocerebellar tracts in paraplegics renders them insensitive to movement of joints, whose associated innervation (articular, cutaneous and muscle) originates below the level of the lesion (Guttmann, 1976).

There has been considerable debate as to which sensory receptors are responsible for kinesthesia (Matthews, 1982). Currently, there seems to be general agreement that muscle and cutaneous receptors are the most likely candidates. Articular receptors of the joint capsule are probably not important for conscious awareness of speed or direction of joint movement since these sensations are not impaired to any appreciable degree when articular receptor activity is lost (Burgess et al., 1982). Muscle spindle endings (Cooper, 1961; Matthews, 1963) and Golgi tendon organs (Alnæs, 1967; Stuart et al., 1970) both respond dynamically to movements which stretch the receptor-bearing muscles. Cutaneous receptors (Hulliger et al., 1979) respond dynamically to movements of nearby joints which affect the stresses and strains in the receptor-bearing tissue.

The extent to which each type of receptor is normally involved in kinesthesia has not been resolved. Their

relative importance is likely to vary with such factors, as receptor density, angular position of the joint with respect to its extremes and parameters of the movement, e.g. amplitude, velocity and acceleration. Body regions with a relatively high density of cutaneous receptors will likely experience a greater contribution by these receptors to kinesthesia than regions where the density is relatively low. Joint angular position will affect muscle length, passive muscle tension and stresses and strains in epidermal tissue. Consequently, it will determine the background discharge rates of stretch receptors, tension receptors and mechanoreceptors, against which dynamic responses must be gauged. Movement amplitude, velocity and acceleration will determine the intensity and duration of these dynamic responses.

Burgess et al. (1982) have pointed out that there are two ways in which receptors related to kinesthesia may function. The first, is in a primary or specific role. The output of receptors acting in this capacity will be correlated with a kinesthetic variable such as velocity, acceleration or force. The second means of action is to facilitate the specific response. When acting in a specific role, sensory receptors provide signals which are sensed as the kinesthetic variables with which they are correlated. Afferent activity originating from sensory receptors acting in a facilitatory role interacts with the primary signals to increase kinesthetic sensitivity.

Whether the action of a receptor is specific or facilitatory is often difficult to assess. For example, kinesthetic sensitivity and intensity are reduced by cutaneous anesthesia (Goodwin et al., 1972; Gandevia and McCloskey, 1976). This may be a specific effect, since cutaneous activity which might be correlated with movement has been eliminated or the effect may be due to the removal of a general facilitation at spinal or higher levels that enhances movement correlated activity of muscle receptors. Cutaneous afferents, in combination with joint afferents, are certainly capable of signaling movement without the action of muscle receptors since kinesthesia persists, albeit in a somewhat cruder form, following disengagement of the muscles to the distal interphalangeal joint of the middle finger (Gandevia and McCloskey, 1976). In the same vein, a facilitatory role for articular receptors need not be ruled out even if their responses are not specific. Receptors may even play more than one role depending on how they are involved in a movement. Cutaneous anesthesia of the adjacent fingers has been shown to reduce movement sensitivity of the middle finger (Gandevia and McCloskey, 1976), suggesting that when the adjacent fingers are stationary, discharge of their cutaneous receptors facilitates the transmission of kinesthetic information up the spinal cord. On the other hand, if the fingers are moving, the discharge of some of the same receptors will likely be correlated with the movement, making their role a

specific one.

The evidence for a specific role for muscle receptors in kinesthesia, particularly primary muscle spindle endings, is compelling. Muscle vibration, which is known to be a potent stimulus to muscle spindle primary endings, will induce an illusion of joint movement in a restrained limb (Goodwin et al., 1972). Although vibration is not a stimulus which is purely selective for primary endings, their activity is more highly modulated than that of any other stimulated receptors (Burke et al., 1976). Moreover, the vibration induced reflex muscle contraction which accompanies the movement illusion, is similar to the tonic vibration reflex seen in cats, which is assumed to be mediated by primary endings (Matthews, 1966; Brown et al., 1967). Evidence against the possibility of Golgi tendon organs being implicated in the illusion comes from observations of the effects of vibration and loading during muscle fatigue. The perceived angular velocity of the illusion decreases with increasing voluntary contraction of the vibrated muscle (Goodwin et al., 1972; McCloskey, 1973). In itself, this does not distinguish between muscle spindle and tendon organ afferents since both would be expected to discharge at higher rates with increasing muscle contraction, the former because of fusimotor effects and the latter because of their sensitivity to actively generated muscle force. However, the fact that muscle fatigue further reduces the perceived velocity for the same loading

(McCloskey, 1973) suggests that the increased discharge due to vibration has become a smaller fraction of the total discharge. This would be expected as a consequence of coactivation of alpha- and gamma-motoneurons if muscle spindle afferents were responsible, but tendon organs should not increase their discharge rates unless active force increases. It appears, therefore, that the movement illusion is indeed the result of excitation of muscle spindle afferents, particularly primary endings.

Since the illusion always appears as one of movement in the direction of stretch of the vibrated muscle, the excess spindle primary afferent discharge is being interpreted as lengthening of the vibrated muscle. Increasing the frequency of vibration, which produces a corresponding one-to-one increase in the rate of spindle primary afferent discharge up to 30-50 Hz, causes the illusory velocity to increase, as well (Roll and Vedel, 1982).

It is possible that during voluntary movements kinesthesia may result from corollary discharges. McCloskey (1981) derives the concept of corollary discharges from the concept of motor commands. A motor command is to be understood as a discharge or pattern of discharge which is generated within the central nervous system and leads to the excitation of spinal motoneurons. All signals that stem from motor commands and that remain entirely within the central nervous system are termed internal command collaterals. When these internal command collaterals affect sensations, they

become corollary discharges.

Corollary discharges may effect a central modification of the processing of sensory signals generated peripherally or they may evoke sensation by their own independent actions. The latter could be thought of as a central 'image' of the movement which is derived from the motor command preceding the movement, rather than being a consequence of the movement. There have been reports that sensations of movement can arise in the absence of peripheral feedback, as for example, in a situation where a digit has been anesthetized and an attempted movement is unexpectedly prevented from occurring (Kelso, 1977; Kelso and Holt, 1980). However, it is doubtful whether such muscle contractions are ever perceived as movements. It is more likely that the subject, realizing that his kinesthetic sensibility is impaired, simply infers that each movement attempt has been successful.

The view that corollary discharges alone do not lead to movement sensation is supported by the observation that in the situation obtaining when both afferent and efferent transmission are severely impaired, movements may be commanded and executed without being perceived (Goodwin et al., 1972). Even stronger evidence is the fact that an attempt to move a paralyzed finger is not perceived as a movement, even when sensory stimuli appropriate for the sensation of movement are provided artificially (McCloskey and Torda, 1975).

The majority of evidence favors the view that internal command collaterals interact with incoming peripheral sensory signals to elicit sensation during voluntary movements. For this reason, illusions and misinterpretations of sensory data would be expected when the normal balance between outgoing and incoming signals is altered. Such effects have been observed and are well-documented. In addition to the illusion of movement induced by muscle vibration, there are illusions of the extent of movement when digits are anesthetized or muscles partially paralyzed (Goodwin et al. 1976). Fel'dman and Latash (1982), in a logical extension of Fel'dman's equilibrium point theory, have shown how kinesthetic illusions could arise if the motor commands which are issued do not result in their intended or expected consequences.

While a motor command for movement may not result in the sensation of movement on its own, the motor command is undoubtedly sensed in a manner which reflects the intensity of the commanded muscle action. Even if movement does not ensue, the subject can distinguish roughly between commands that would have resulted in relatively faster or slower movements. The question then arises as to whether this sense of effort is simply a consequence of a peripheral sensory signal related to force or whether it arises independently of afferent feedback. Gandevia (1982) concludes that afferent input is not essential for producing sense of effort since this sensation persists after dorsal rhizotomy

or spinal transection. That sense of effort is causally related to the motor command has been demonstrated by the observation that patients with paralysis resulting from hemiplegia, without accompanying sensory symptoms, sense no effort when attempting to contract completely paralyzed muscles, i.e. when no motor command is issued there is no sensation of effort. Only when paralysis is incomplete is effort sensed.

Although sense of effort is related to the strength of the motor command rather than the resulting muscle force (McCloskey et al., 1974; Gandevia and McCloskey, 1977a,1977b; Cafarelli and Bigland-Ritchie, 1979), by itself, sense of effort does not appear to provide a long-lasting, reliable estimate of muscle activation. This is suggested by the inability of a deafferented patient to maintain a prescribed joint torque (Rothwell et al., 1982). In this case electromyograms were not shown so it is not possible to ascertain whether changes in joint torque were accompanied by changes in the balance of activity between antagonist or synergistic muscles which may have still been sensed as the same effort. As Gandevia (1982) noted, in the absence of afferent input to the motor centers, it may be difficult to focus signals of effort to activate the appropriate muscles.

Kinesthesia involves the ability to perceive relative movement velocities. As noted above, muscle spindle afferents have been implicated in this regard, particularly

for movements of slow, constant angular velocity such as those of the vibration induced illusion (Roll and Vedel, 1982). However, any attempt to unequivocally assign the function of velocity sensation to muscle spindle afferents is beset with complications. First, the responses to stretch are highly nonlinear, involving a transient at stretch onset (Lennerstrand and Thoden, 1968; Brown et al., 1969) followed by activity which is dependent on both muscle length and stretch velocity (Houk et al., 1981). Secondly, an increase in spindle afferent discharge can depend equally on muscle stretch or fusimotor activity, but fusimotor actions do not induce movement illusions (Vallbo et al., 1979; Hulliger and Vallbo, 1979; Hulliger et al., 1982). For muscle spindle afferents to function as reliable velocity sensors, their output would have to undergo considerable processing, particularly in reference to the efferent command to gamma-motoneurons, before reaching consciousness. Houk et al. (1981) have suggested that muscle spindle receptors may be better suited for movement detection than for signaling the precise velocity at which movement occurs.

Even though muscle spindle afferent activity has been shown to be correlated with velocity for constant velocity stretches, it is probably not a very sensitive measure of instantaneous velocity during rapid movements in which angular velocity and hence, stretch velocity is never constant. For such movements, average velocity or peak velocity would be a more appropriate measure of movement

speed, given the limitations in sensitivity of sensory receptors. Of the two, peak velocity would more adequately characterize the initial acceleration associated with rapid movements. However, there is still ambiguity in the response, which must be resolved by higher level processing. As Hagbarth et al. (1975) have shown, muscle spindle afferents discharge with bursts of activity both during the active shortening and passive lengthening phases of rapid movements, but in this case the signal may be easily resolved since, even though agonist and antagonist spindle activity might coincide, both would be related to the initial acceleration. Resolution would simply be a matter of determining the direction of movement.

E. Objectives

The present study was undertaken to gain a better understanding of some of the processes involved in the regulation and perception of velocity in simple, preprogrammed movements. Brooks and Thach (1981) classified these movements in two categories: 'ballistic' movements which involved no active braking action by antagonist muscles and 'self-terminated' movements which did. 'Ballistic' rather than 'self-terminated' movements were chosen for this study in order to extend the range of velocities that could be achieved and to provide a more direct causal link between agonist muscle activity and movement velocity.

As noted above, the initial acceleration of 'self-terminated' movements is regulated by varying the amplitude of the initial agonist burst, while keeping its duration constant. The agonist activity is correlated with both movement amplitude and velocity (Bouisset and Lestienne, 1974; Lestienne, 1979; Hallett and Marsden, 1979; Cooke and Brown, 1981; Marsden et al., 1983). It has also been reported that such movements exhibit a linear relationship between movement amplitude and peak angular velocity (Bouisset and Lestienne, 1974; Wadman et al., 1979; Ghez, 1979; Cooke, 1980; Georgopoulos et al., 1983b) suggesting that initial acceleration and movement amplitude may both be parameters used in the regulation of peak angular velocity.

The first part of this investigation examines how these two parameters interact in the regulation of peak angular velocity during 'ballistic' movements. It was hoped that by imposing constraints on movement amplitude and velocity, it would be possible to identify movement strategies and gain some insight into the flexibility of the central nervous system's capabilities in programming simple movements.

The remainder of the study focuses on the role that afferent input plays in the programming process. 'Ballistic' movements are executed in too short a period to be continuously regulated. Therefore, the motor command cannot be updated by peripheral sensory feedback. Judgments based on sensory feedback can only be made following execution of

the movement. To be successful in attaining the objective (in this case the achievement of a prescribed peak angular velocity), the programming centers must adapt the programmed motor command to suit the prevailing state of the internal and external environments. That is, they must take into account the state of the actuator (the target muscles and their motoneuron pool) and the external forces opposing motion. This involves an assessment of relevant afferent feedback. If conditions change, the programmed motor command must be modified accordingly.

The programming centers would be expected to evaluate sensory information generated by the movement in making an assessment of how successfully the movement met the objective. Whether or not peak angular velocity is actually sensed, some aspect of kinesthesia must be taken as a relevant standard for an assessment of peak velocity. The chosen sensation, which may arise from corollary discharges, as well as from peripheral sensory feedback, must be graded with peak angular velocity if judgments of relative movement speed are to be possible.

This study attempts to establish the criteria used by the programming centers for assessing the success of a motor command in producing a prescribed peak angular velocity. It focuses on misjudgments brought about by changes in the state of the internal and external environments. These changes in state were induced experimentally by varying the initial muscle length and the force opposing movement.

II. GENERAL PROCEDURE

A total of 14 male and female volunteers between the ages of 19 and 56 participated in this study. Subjects gave informed consent to the experimental procedures. I was a subject in all experiments.

Movements of the interphalangeal joint of the right thumb were studied. Subjects sat in a chair of adjustable height, raised to a level which allowed the forearm to rest comfortably on a table. The right forearm and hand were rigidly supported at the elbow and wrist to prevent lateral movement. The proximal phalanx of the right thumb was firmly clamped so that movement of the distal phalanx could be effected only by contractions of the extensor pollicis longus (EPL) and flexor pollicis longus (FPL) muscles. These are the only muscles whose tendons are attached to the distal phalanx of the thumb. The end of the thumb was clamped in a light cage which rotated in a vertical plane about an axis passing through the center of rotation of the interphalangeal joint of the thumb (Figure 4).

Joint angle or angular position of the thumb was measured using a linear potentiometer attached to a shaft along the axis of rotation. Angular position was taken as zero when the cage was horizontal. This was the position in which the EPL and FPL muscles were most relaxed, i.e. where e.m.g. activity was minimal when the proximal phalanx of the thumb was supported in the clamp.
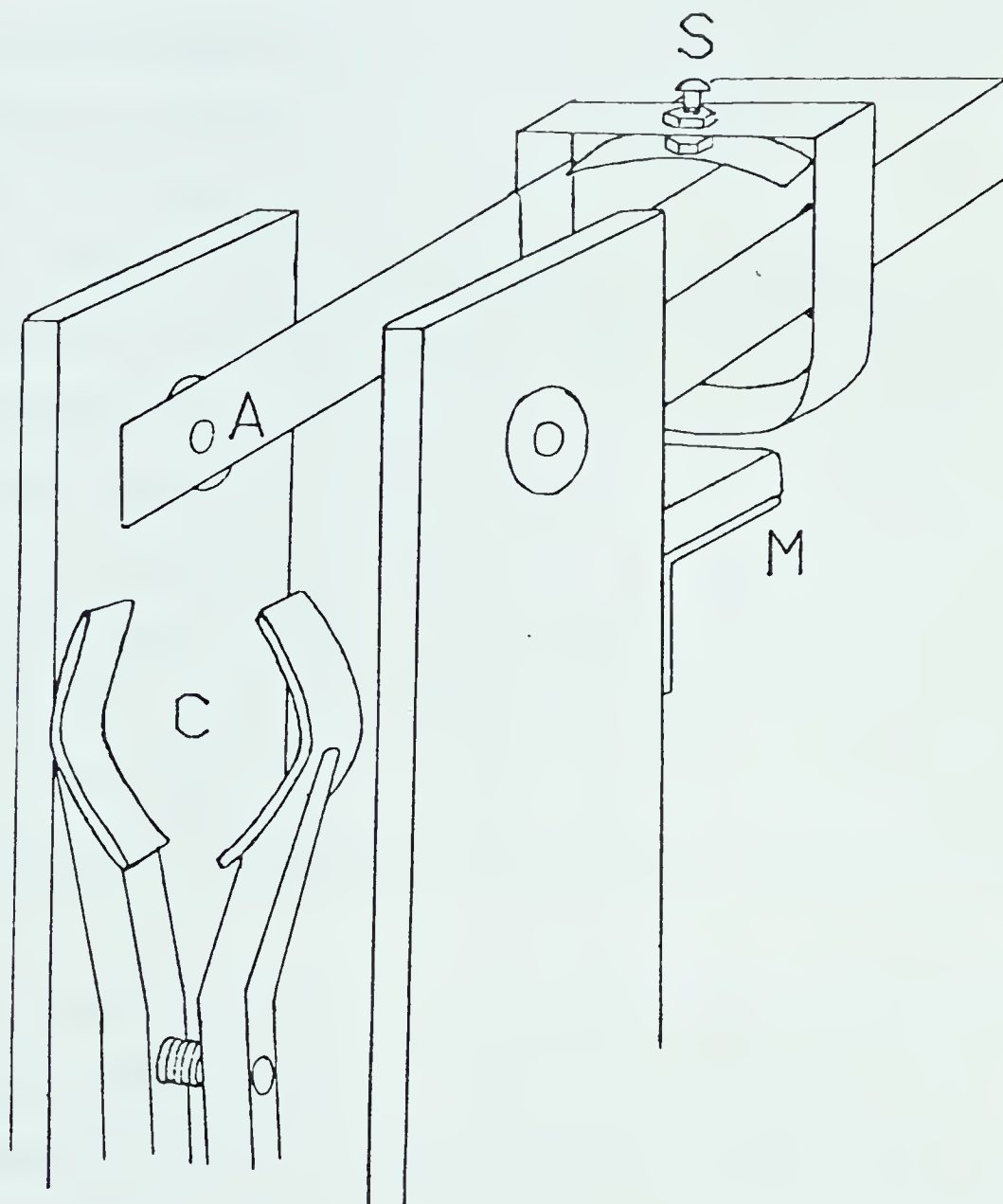
FIGURE 4. The experimental apparatus consisted of a light metal cage in which the distal phalanx of the thumb was secured by tightening the screw (S). The thumb was positioned to align the interphalaneal joint with the axis of rotation of the cage (A). The proximal phalanx was held in a clamp (C) to constrain movement to the interphalangeal joint. A mechanical stop (M) limited flexion.

Joint angular velocity was obtained from the output of a moving-coil galvanometer which rotated on the same axis as the potentiometer. Position and velocity signals were low-pass filtered with a cutoff at approximately 100 Hz. They were then amplified and were either recorded on tape or digitized and stored on disc, together with the e.m.g.'s obtained from EPL and FPL muscles.

The e.m.g. from the FPL muscle was generally recorded using a pair of silver-chloride discs 1 cm in diameter which were attached to the skin overlying the distal aspect of the muscle. EPL e.m.g. was recorded in a similar fashion using a pair of silver-chloride discs 3 mm in diameter. The smaller discs were used for EPL e.m.g. because less muscle area was accessible for recording. The electrodes were positioned to maximize the amplitude of e.m.g. bursts recorded during brisk flexion or extension of the distal phalanx of the thumb.

On occasion, e.m.g. was recorded using concentric electrodes consisting of a 1 cm silver-chloride disc surrounded by a flat ring cut from 0.0125 mm brass shim stock. The inner diameter of the ring was approximately 2 cm and its width approximately 3 mm. These electrodes were more difficult to use and were less sensitive than the conventional electrodes, but they proved to be somewhat more selective in restricting signal pick-up to the closest muscles. Generally though, there was little evidence of cross-talk in records obtained from EPL and FPL muscles when

using conventional electrodes.

E.m.g.'s were fed into a preamplifier with a high-pass cutoff of 10 Hz and subsequently amplified at a second stage before being recorded on tape or digitized for disc storage. If e.m.g. was to be digitized, it was rectified at the second stage of amplification.

When the four data channels were averaged on-line they were sampled at 2 kHz. When they were sampled as continuous records either directly or from tape, the sampling rate was reduced to 1.67 kHz. With the exception of one experiment, the e.m.g.'s were not low-pass filtered before being digitized. Only in the first experiment described in the next chapter were the e.m.g.'s filtered. A Paynter filter with a low-pass cutoff of 100 Hz was used and the sampling rate was reduced to 500 Hz. All digitized e.m.g. records were subsequently analyzed using programs written for a PDP11/34 or LSI11 computer. At this stage, e.m.g. records were smoothed using software implementation of a finite impulse response filter with a low-pass cutoff of 10 Hz.

All voluntary movements of this study brought the rotating cage of Figure 4 into contact with a mechanical stop at their lowest extent. The stop limited flexion to an angle of approximately zero radians as measured from the horizontal. This angle was taken as zero radians of extension. Thus, angular position increased with extension and decreased with flexion. The surface of the stop was padded to cushion the impact of the cage. This reduced the

amplitude of vibrations transmitted to the galvanometer and
attenuated stimuli during impact which were potential
sensory cues for movement reproduction.

III. RHYTHMIC RECIPROCATING MOVEMENTS

A. Methods

I was the only subject of the initial experiment which was undertaken as a quantitative study of the relationship between movement amplitude, muscle activity and peak angular velocity of movement. The task was to perform a sequence of reciprocating movements in rapid succession. Each sequence consisted of 11 movements which followed a strict temporal pattern. The object of each movement was to attain a peak angular flexion velocity which fell into one of three specified target ranges using the sequence 1,1,1,2,3,2,1,3,2,2,1 (velocity increasing with target number).

Seven sequences were performed, each at a slightly faster tempo than the previous one. The slowest tempo had an average movement frequency of 2.2 Hz, the fastest 4.8 Hz. The task proved to be too complex to be executed accurately, particularly when the average movement frequency exceeded 4 Hz. It did, however, provide useful data regarding the regulation of velocity and stimulated a more systematic investigation which is described in the next chapter.

B. Results

Figure 5 illustrates a typical 'ballistic' reciprocating movement. The e.m.g.'s of the EPL and FPL muscles are characterized by brief bursts of activity
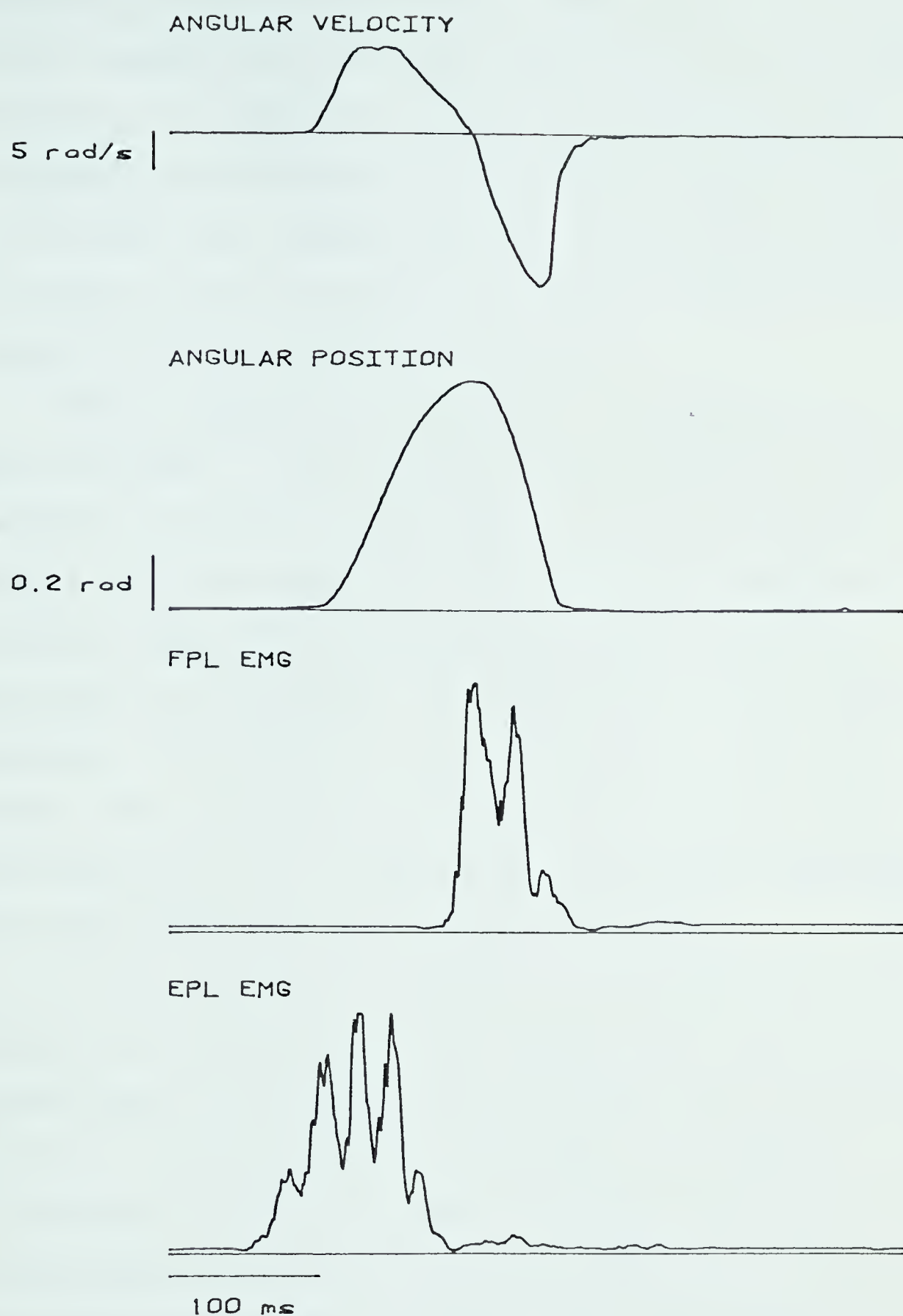
FIGURE 5. Typical reciprocating movement. Extension (increase in angular position) was immediately followed by flexion (decrease in angular position). There is no overlap between the e.m.g. bursts in EPL and FPL. Note that the FPL e.m.g. burst begins less than 25 ms prior to the onset of flexion.

preceding extension and flexion respectively. The baseline of the angular position trace represents the zero angle of extension; the baseline of the angular velocity trace represents zero velocity. As the distal phalanx of the thumb is extended, the angular position rises. Movement amplitude is taken as the peak angle of extension. Flexion begins when angular velocity crosses the baseline.

When a sequence of 'ballistic' reciprocating movements was performed so that the peak flexion velocity varied from movement to movement (equivalent to varying the force of impact with the mechanical stop), velocity covaried with movement amplitude and intensity of muscle activity (Bouisset and Lestienne, 1974; Lestienne, 1979; Hallett and Marsden, 1979; Wadman et al., 1979; Ghez, 1979; Brown and Cooke, 1981; Hoffman and Strick, 1982; Marsden et al., 1983; Georgopoulos et al., 1983b). This observation applied to both peak extension and flexion velocities.

In the analysis of sequential movements, the time between the initiation of one movement and the next was separated into two parts. The first part was the movement duration, lasting from the initiation of extension to the termination of flexion. These two events were quite clear from records of angular velocity (Figure 6). The second part was the inter-movement interval, the time between the termination of flexion for one movement and the initiation of extension for the next. This interval appears as the flat region in records of angular velocity. The average frequency

ANGULAR VELOCITY

5 rad/s
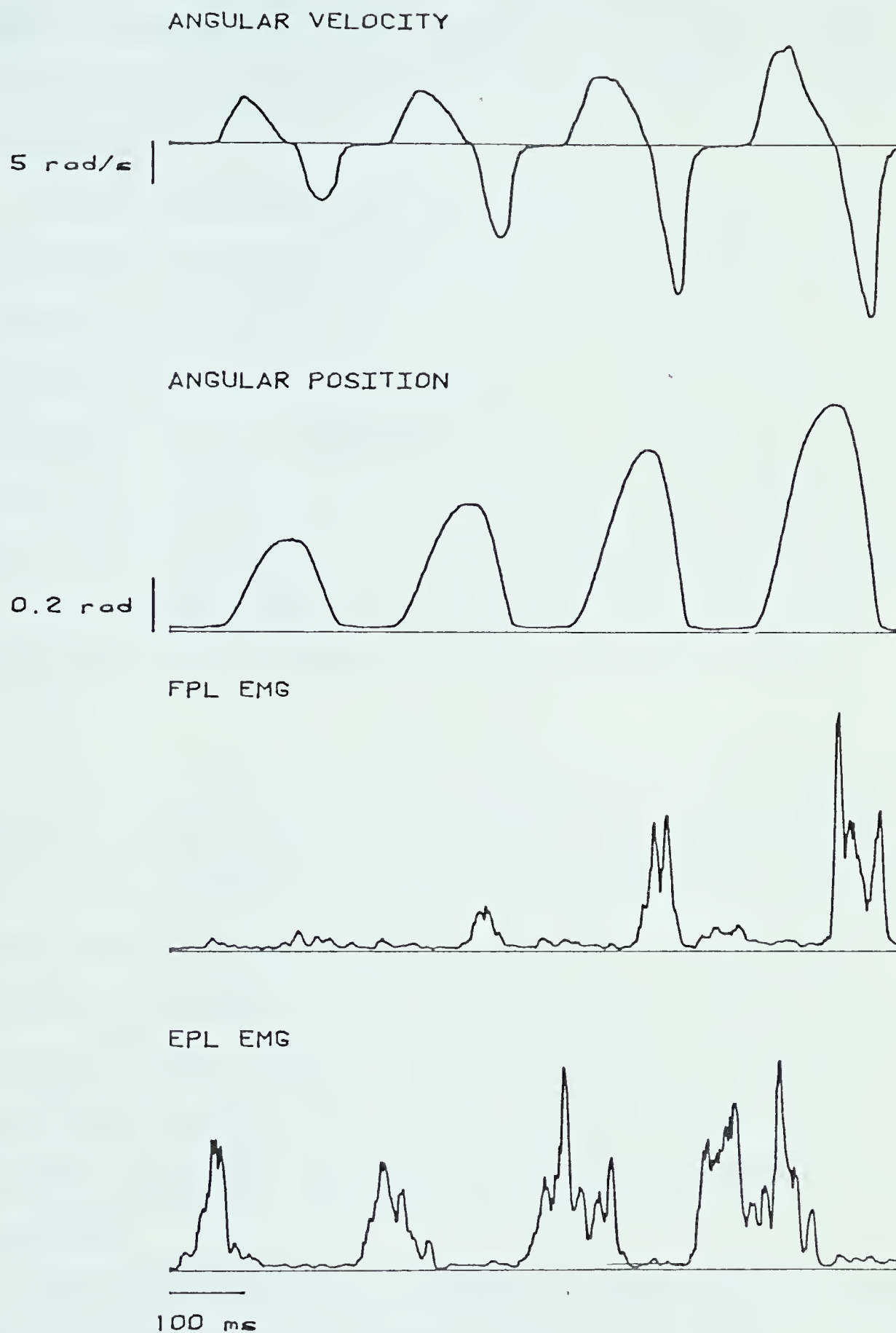
ANGULAR POSITION

0.2 rad

FPL EMG

EPL EMG

100 ms

FIGURE 6. This series of reciprocating movements illustrates that peak velocity covaries with movement amplitude and muscle activity.

of a sequence of movements was determined by dividing the number of movements in the sequence by the time between termination of flexion in the first and last movements of the sequence.

Linear regression analysis was carried out on the pooled data of seven movement sequences(average movement frequency ranging from 2.2 to 4.8 Hz). There were strong correlations between peak extension velocity and movement amplitude (r=0.71), and peak flexion velocity and movement amplitude (r=0.87), as well as between the integrated EPL e.m.g. burst amplitude or area and peak extension velocity (r=0.70, r=0.82), and the integrated FPL e.m.g. burst amplitude or area and peak flexion velocity (r=0.76, r=0.82).

When movement sequences were analyzed individually, similar correlations were obtained within each sequence when the average movement was below 4 Hz. Above 4 Hz the ordered relationship between movement amplitude, e.m.g. burst intensity and peak velocity sometimes broke down, weakening the correlation. Figure 7 shows sequential movements in which peak flexion velocity was nearly the same, but movement amplitude and FPL e.m.g. intensity varied inversely.

The second to fifth movements of each eleven movement sequence encompassed a range of peak velocities, but were approximately equal in duration for a given sequence (Figure 8). It was, therefore, possible to examine the relationships
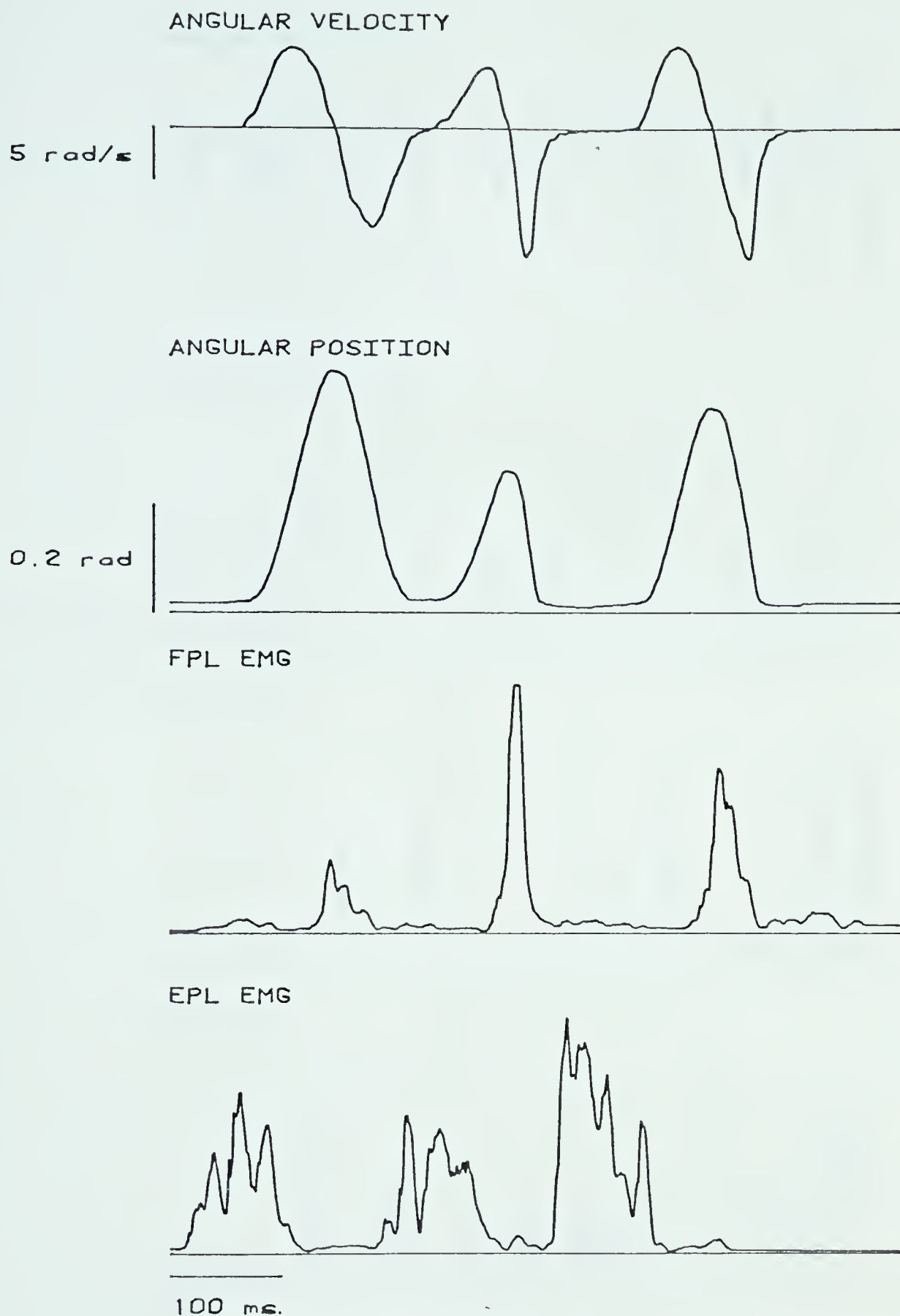
FIGURE 7. Disordering of the relationship between amplitude, velocity and e.m.g. at fast tempos. First movement has greater amplitude but lower peak flexion velocity than the other two. Second movement has lower amplitude than third but approximately same peak flexion velocity. This inverted relationship between between amplitude and velocity (with respect to Figure 6) is accompanied by a corresponding inverted relationship between amplitude and FPL e.m.g.

ANGULAR VELOCITY

5 rad/s

ANGULAR POSITION

0.2 rad

FPL EMG

EPL EMG

200 ms
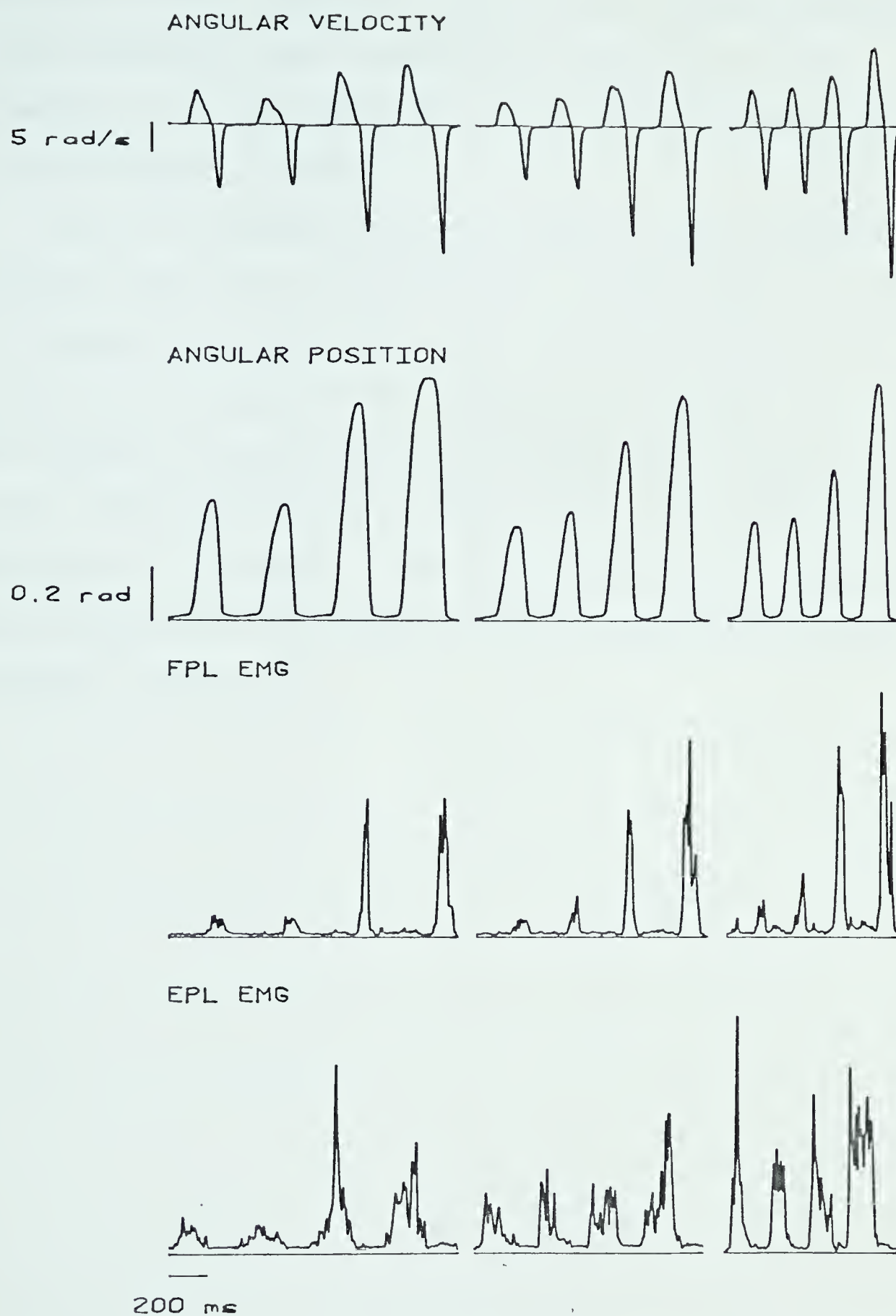
FIGURE 8. Second, third, fourth and fifth movements of three 11 movement sequences (average frequencies: 2.5 Hz, 3.2 Hz and 4.4 Hz). Movement duration, inter-movement interval and e.m.g. burst durations all decrease as the tempo increases.

between movement frequency and various timing parameters. These parameters were averaged for each set of four movements and correlated with their average frequency. The results appear in Table 1.

Both the movement duration and the inter-movement interval were highly correlated as inverse power functions of frequency ($r > 0.99$). The inter-movement interval decreased faster with frequency than the movement duration (Figure 9A) as Stetson and McDill (1923) had observed in their early study. The durations of the EPL burst and the FPL burst also decreased with movement frequency, EPL duration dropping more rapidly than FPL duration which was relatively more constant (Figure 9B).

TABLE 1

Velocity, Amplitude and E.M.G. of Rhythmic Movements
(linear regression on pooled data from 7 sequences)

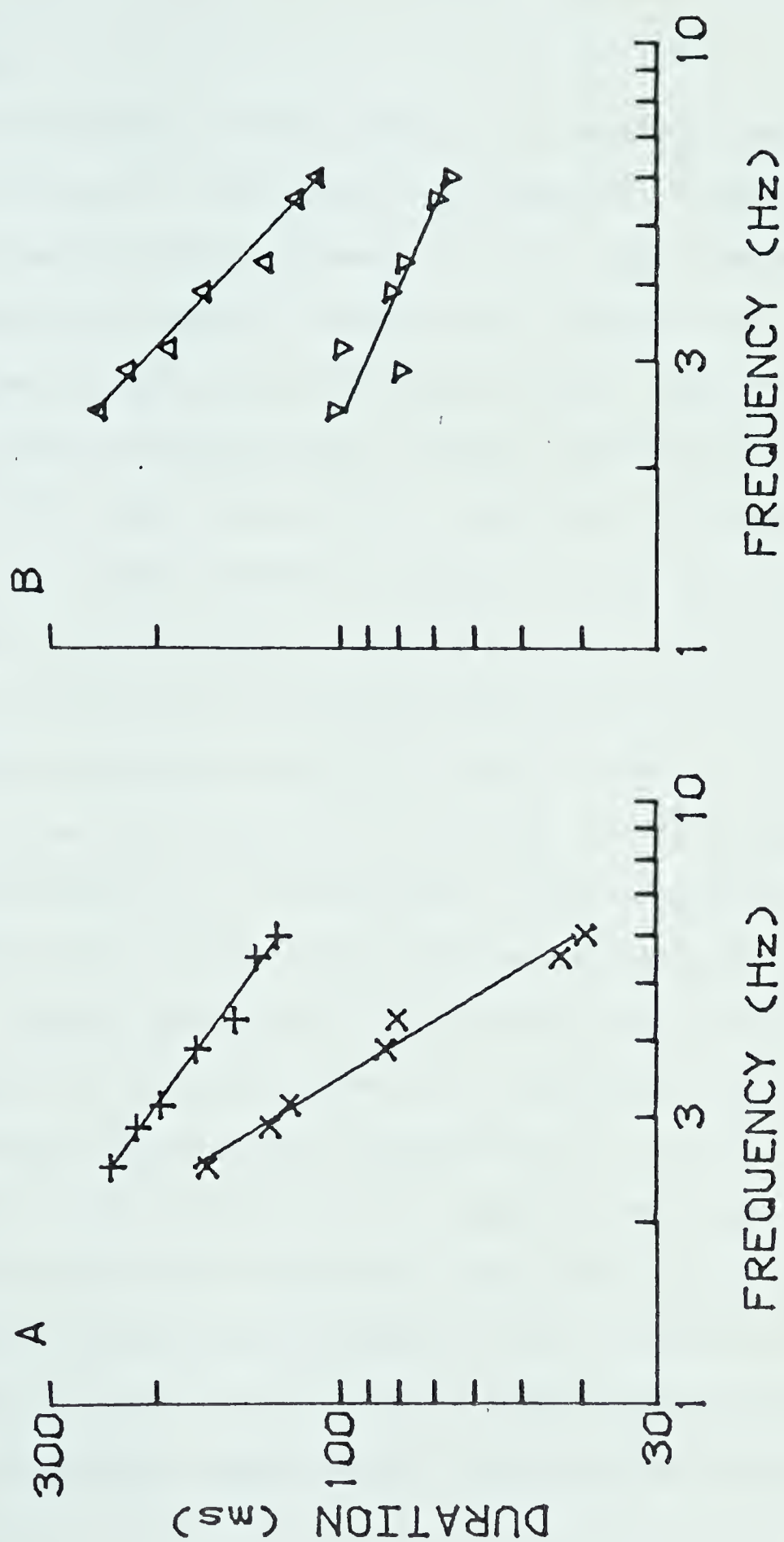| Correlated Parameters	Slope[s.e.](n=77)	r
Peak Extension Velocity vs Amplitude	9.23[1.06]	0.71
Peak Flexion Velocity vs Amplitude	20.88[1.35]	0.87
Peak Extension Velocity vs Integrated EPL E.M.G.	0.35[0.03]	0.82
Peak Flexion Velocity vs Integrated FPL E.M.G.	1.06[0.08]	0.82
Peak Extension Velocity vs EPL E.M.G. Duration	-0.017[0.004]	0.43
Peak Flexion Velocity vs FPL E.M.G. Duration	0.032[0.021]	0.17

FIGURE 9. A. Log-log plots of movement duration (+) and inter-movement interval (X) versus frequency. B. Log-log plots of EPL e.m.g. burst duration (▲) and FPL e.m.g. burst duration (▽) versus movement frequency. Plotted points are averages obtained from four movements. Linear regression coefficients are listed in Table 1.

IV. SINGLE RECIPROCATING MOVEMENTS

A. Methods

To investigate in more detail the strategy used to control 'ballistic' reciprocating movements, subjects were asked to execute single movements rather than the sequences of movements described in the previous chapter. Six subjects participated in this series of experiments. They each performed three tasks during a single recording session.

The first task involved the regulation of movement amplitude, i.e. peak extension angle, the second task, the regulation of peak angular flexion velocity and the third task, the regulation of both amplitude and velocity.

Subjects were provided with visual feedback of the movements from the display of a storage oscilloscope. The display consisted of a fixed target window delimited by two bright horizontal lines, along with an instantaneous display of either angular position, angular velocity or both. Angular position was displayed by a bright dot wich moved upward during extension and downward during flexion. The dot was located at the bottom of the display when the thumb was positioned against the mechanical stop. The persistence of the displayed trace was adjusted so that movement of the dot left a visible trail which quickly faded following movement, but gave the subject opportunity to assess the accuracy of the movement.

A second dot displayed angular velocity, moving upward during extension and downward during flexion. The dot was located at the top edge of the display when the velocity was zero. Thus, only flexion velocity could be seen by the subject.

In the first task only angular position was displayed; in the second, only flexion velocity; in the third, both angular position and flexion velocity were displayed.

It was not necessary to hide the hand from the subject's view since there were no noticeable differences between results obtained when the hand was visible and when it was not.

In the first task, subjects were instructed to make single 'ballistic' reciprocating movements of an amplitude which constrained the position dot to be in the target zone at the point of maximal extension. They were told to make each movement rapidly and accurately, but to separate individual movements by several seconds. They were presented with four to six targets ranging in amplitude from 0.15 to 1.00 rad.

In the second task, subjects were given similar instructions, but were now required to get the velocity dot into the target zone at its point of maximal downward excursion during flexion. They were presented with four to six targets ranging from 5 to 34 rad/s.

In these tasks, two subjects were presented with a target zone whose location on the oscilloscope screen varied

while the position or velocity gains remained fixed. Thus, in order to enter the target zone, the position or velocity dot had to be moved greater distances as the magnitude of the target position or velocity was increased. For the other subjects, the position or velocity gains were varied to keep the target zone in the same location on the display. The results obtained under conditions of fixed or variable gain did not differ.

In the third task, three subjects were presented with a single target for movement amplitude along with one of four (or in one instance, five) targets for peak flexion velocity. The other three subjects were given a single target for flexion velocity along with one of four targets for movement amplitude. The fixed target amplitudes or velocities were chosen to be approximately midway between the extreme ranges of the first and second tasks. Position and velocity gains were adjusted so that in each case position and velocity target zones coincided. The subjects were instructed to make 'ballistic' reciprocating movements which put both dots in the target zone.

Linear regression analysis was used to correlate peak angular extension velocity, peak angular flexion velocity and movement duration with movement amplitude. All movements, with the exception of aborted attempts or movements with particularly long durations, were included. To quantify the relationship between parameters of the e.m.g. and parameters of the movement in the first two

tasks, records of angular velocity, angular position and e.m.g. were averaged. Five movements were chosen from among all of the movements made to each target (generally 20 to 30). The choice was based on two criteria: the first being that the target parameter (movement amplitude or peak flexion velocity) be within approximately 5% of the mean of all movements made to that target; the second, that all five movements have approximately the same duration.

To check that this procedure provided a representative sample, the regression coefficients for the relationships between movement parameters for the whole population and the selected sample were compared and found to be similar. In the third task, five-movement averages did not provide a representative sample and hence were not used.

B. Results

There were strong linear correlations between peak extension velocity and movement amplitude $(0.72 \leq r \leq 0.98)$ and peak flexion velocity and movement amplitude $(0.88 \leq r \leq 0.97)$ for all subjects in both the amplitude and velocity tasks (Bouisset and Lestienne, 1974; Wadman et al., 1979; Ghez, 1979; Cooke, 1980; Georgopoulos et al., 1983b).

Figures 10 and 11 compare plots of peak extension velocity versus amplitude and peak flexion velocity versus amplitude for the three tasks. In Figure 10C the velocity target was fixed while the amplitude target varied; in Figure 11C the amplitude target was fixed while the velocity

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FIGURE 10: Plots of peak extension velocity and peak flexion velocity versus amplitude for one subject in three tasks. A) Amplitude task: regulating peak angle of extension. B) Velocity task: regulating peak flexion velocity. C) Amplitude + velocity task: regulating amplitude while keeping peak flexion velocity fixed.
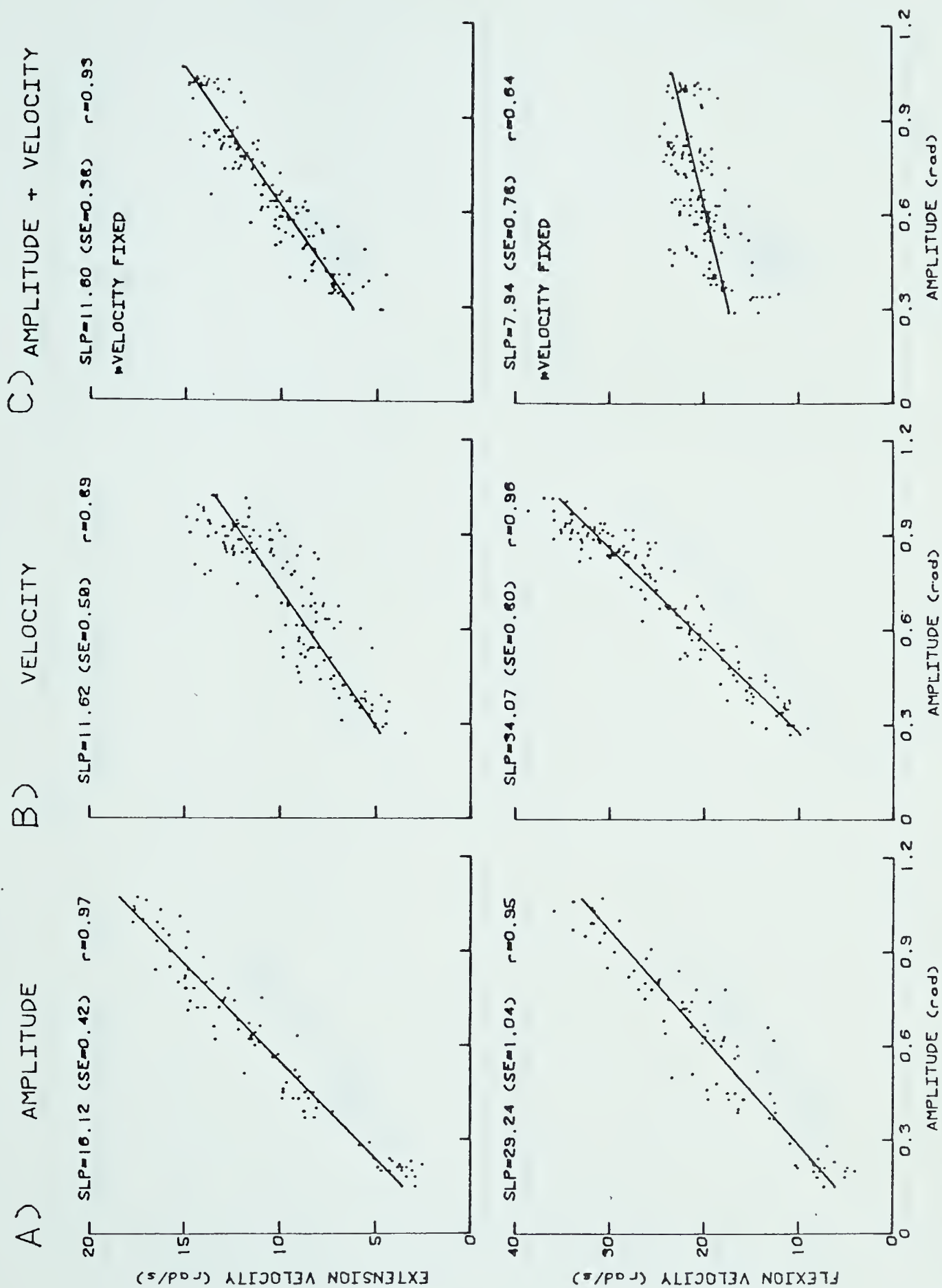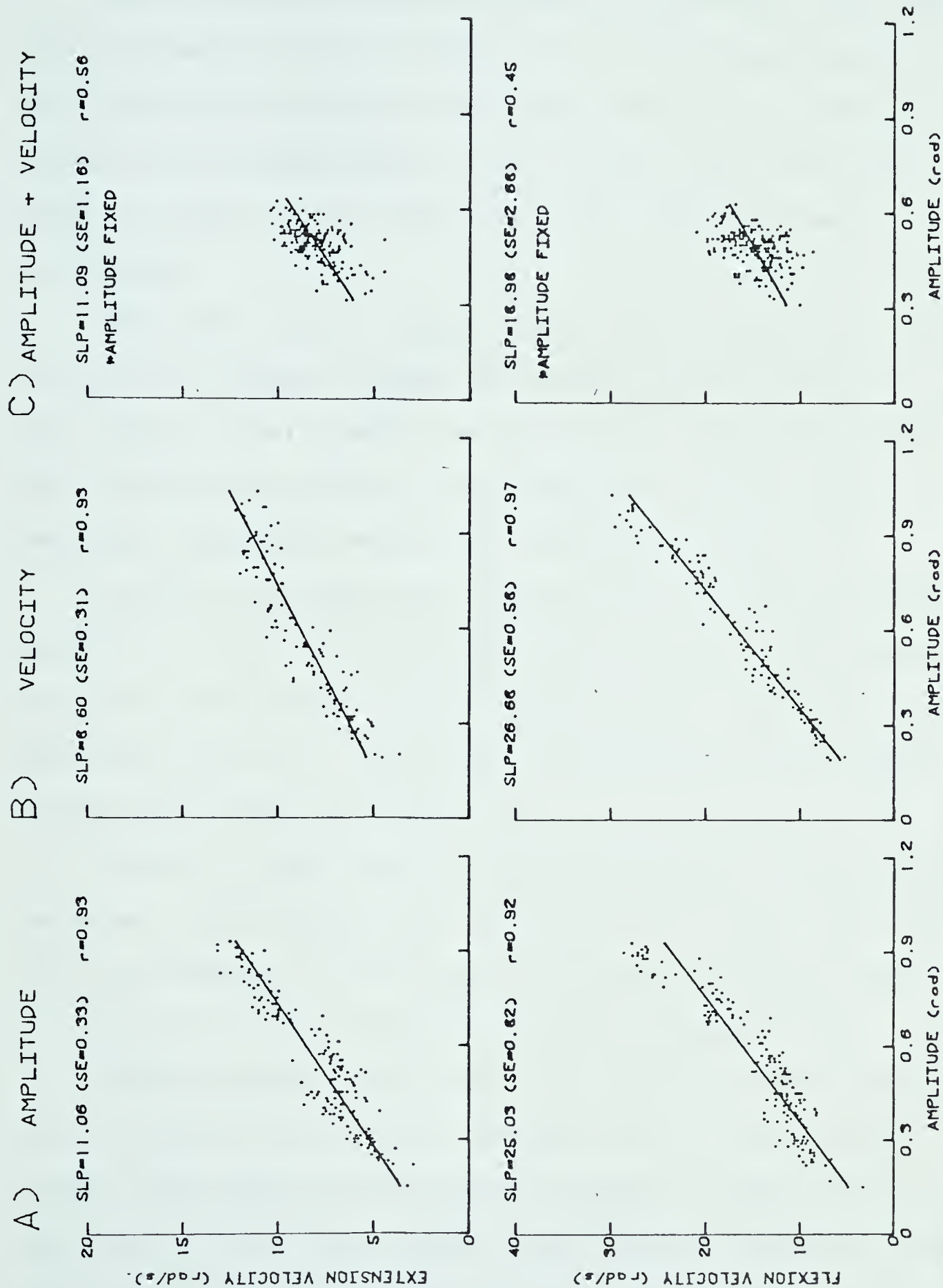
FIGURE 11. Plots of peak extension velocity and peak flexion velocity versus amplitude for another subject in three tasks. A) Amplitude task: regulating peak angle of extension. B) Velocity task: regulating peak flexion velocity. C) Amplitude + velocity task: regulating peak flexion velocity while keeping amplitude fixed.

target varied.

From Figures 10A&B and 11A&B it is evident that there was a tendency for the slope of the relationship between peak extension velocity versus amplitude to be reduced in going from the amplitude to the velocity task while the slope of peak flexion velocity versus amplitude was increased.

When the velocity target was fixed in the third task there was a dramatic reduction in the linear correlation and the slope of peak flexion velocity versus amplitude while the relationship between peak extension velocity and amplitude remained relatively unchanged (Figure 10).

Keeping the amplitude target fixed produced a marked reduction of the linear correlations of both peak extension velocity versus amplitude and peak flexion velocity versus amplitude. However, there was little change in the slope of these relationships (Figure 11).

Figures 12A&B compare angular position trajectories obtained from five-movement averages of the amplitude task for two subjects. In each case there was a strong linear correlation between movement duration and amplitude.

The movements trajectories of the one subject can be seen to deviate soon after movement onset (Figure 12A), larger amplitude movements having greater slopes. The movements of the other subject sometimes followed the same trajectory for much longer periods of time (Figure 12B), particularly as the movement amplitude increased.

A) ANGULAR POSITION

0.2 rad

B) ANGULAR POSITION

0.2 rad

100 ms
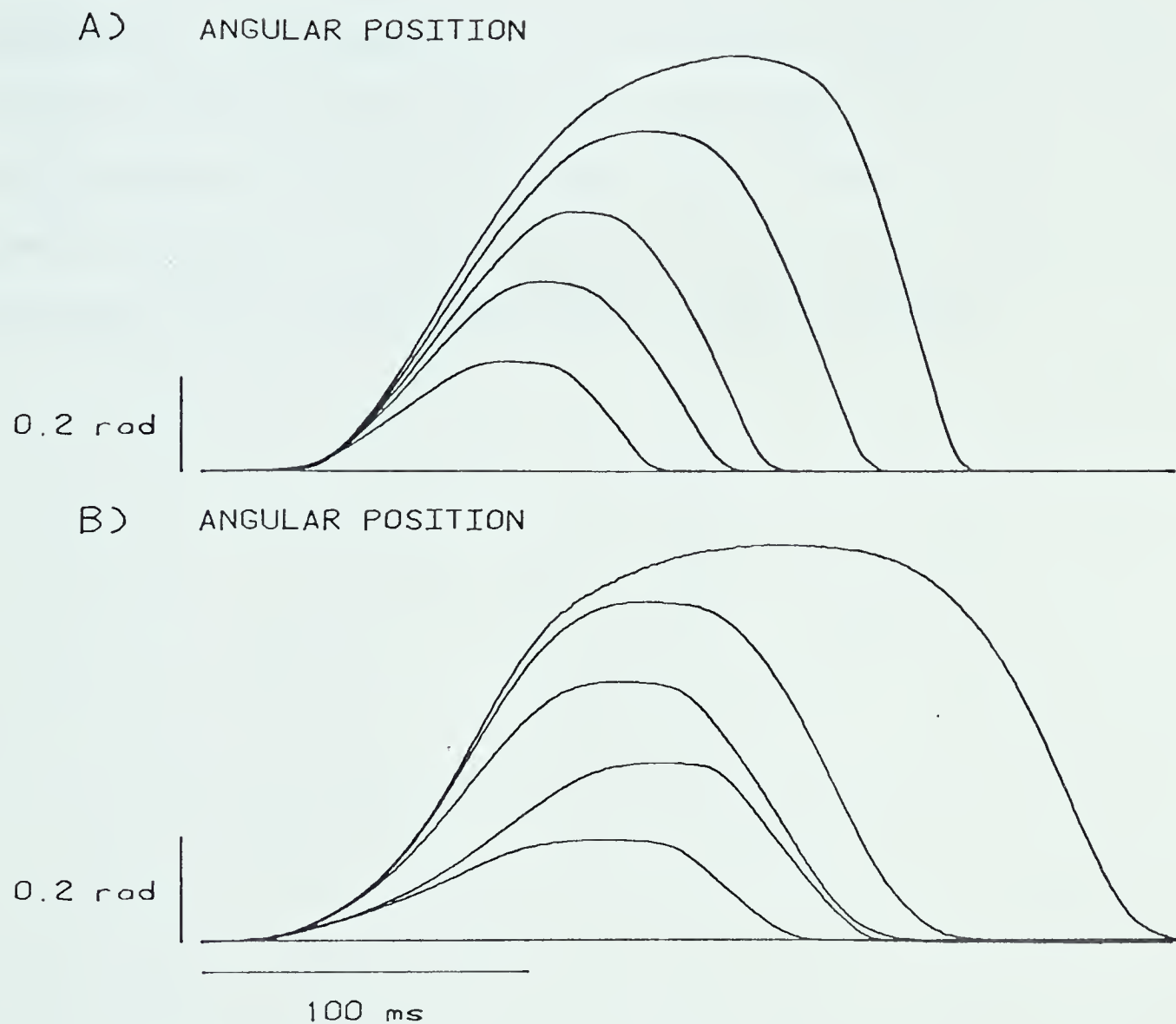
FIGURE 12. Examples of scaled movements from two subjects.
A) Trajectories of increasing amplitude are correspondingly
scaled in duration. B) The two trajectories of least
amplitude and the next two trajectories form pairs which are
correspondingly scaled in time. The trajectory of largest
amplitude appears to be a scaled version of the first pair
as opposed to the second pair.

Figures 13 and 14 show the linear regression lines obtained for the relationships between various e.m.g. parameters and movement parameters for one representative subject. In general, the correlations between e.m.g. burst amplitude or integrated area and movement amplitude or peak velocity were stronger than the correlations between e.m.g. burst duration and movement amplitude or peak velocity, but there was often a strong trend for e.m.g. burst duration to increase with movement amplitude or peak velocity.

FIGURE 13. A) Plots of EPL e.m.g. amplitude, integrated area and duration vs movement amplitude. B) Plots of EPL e.m.g. amplitude, integrated area and duration vs peak extension velocity. All plots are for the same subject.
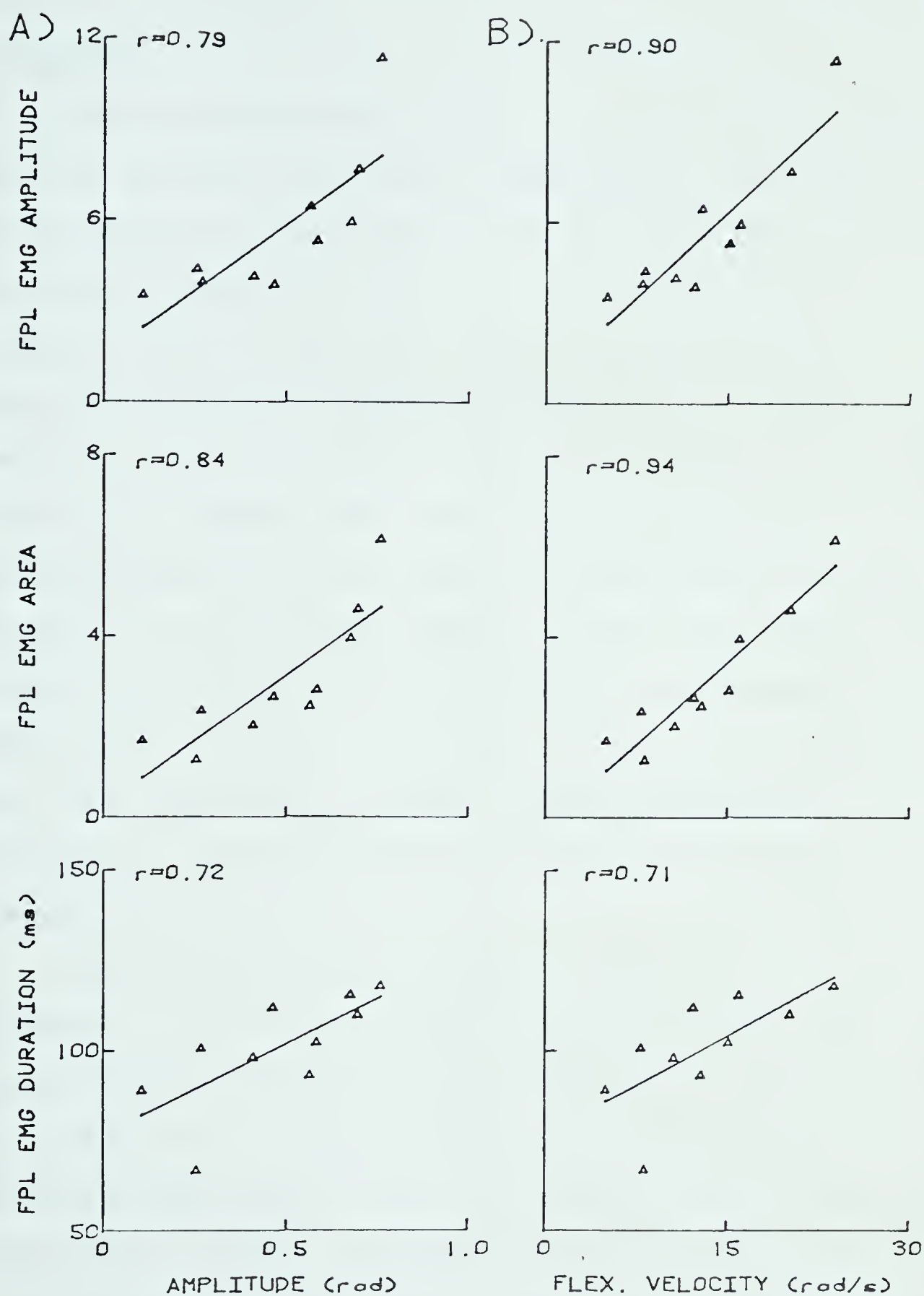
FIGURE 14. A) Plots of FPL e.m.g. amplitude, integrated area and duration vs movement amplitude. B) Plots of FPL e.m.g amplitude, integrated area and duration vs peak flexion velocity. All plots are for the same subject as Figure 13.
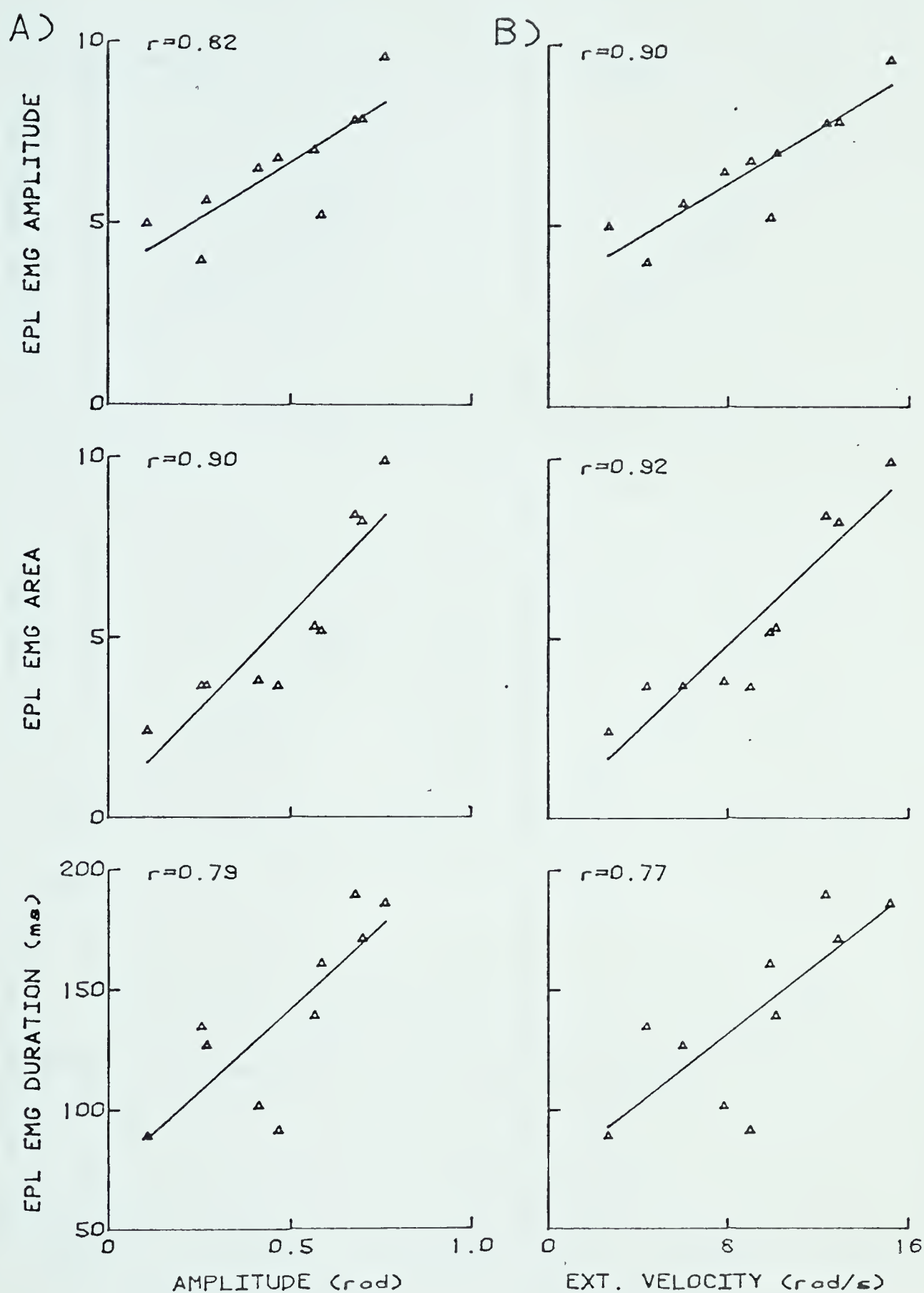
V. SINGLE FLEXION MOVEMENTS

A. Methods

The strength of the linear correlation between peak flexion velocity and movement amplitude in 'ballistic' reciprocating movements and the difficulty experienced by subjects attempting to regulate either parameter independently of the other, suggested that movement amplitude might not only be involved in the regulation of velocity, but also in the judgment of velocity. A similar correlation between peak flexion velocity and the size of the integrated FPL e.m.g. burst intimated that it might be linked to perception of velocity through the sensation of effort experienced when the FPL muscle was voluntarily activated. To further investigate these possibilities, the task was simplified, a visual feedback display was constructed and the method of data acquisition was streamlined.

The modified task consisted of 'ballistic' flexion movements which were initiated after the subject had positioned the thumb at a specified angle of extension. In a few experiments a mechanical stop was used to limit extension and subjects were instructed to begin flexion from this limit. In most experiments though, extension was unobstructed. A visual display provided the subject with information about the angular position of the thumb. The display is described in detail later in this chapter.

A second visual display consisting of a column of red LED's was constructed to signal peak angular velocity. A sample-and-hold peak detection circuit was designed to sample angular flexion velocity. Its output was fed into a comparator circuit where it was compared to ten different voltage thresholds. Voltage steps between successive thresholds were equal, creating nine voltage windows of equal width. The output from each of the ten comparators was applied to a separate LED. An LED would light up in response to an input voltage which exceeded the comparator threshold which it was monitoring, but which fell below the next threshold level. Peak flexion velocity was displayed as a brief flash (approximately 250 ms in duration) of the LED corresponding to the voltage window into which the velocity signal fell.

The circuit was wired so that an LED progressively farther down the column was activated as the magnitude of peak flexion velocity increased. When the magnitude of the velocity increased monotonically until achieving its maximum, only one peak was detected and hence only one LED flashed during flexion. However, the velocity-time profile was often irregular at lower velocities, triggering more than one LED. To prevent this, a lock-out circuit was employed to deactivate the LED display until the movement had crossed an angular position threshold near the stop. This ensured that only the absolute peak of the velocity activated an LED.

The velocity display could be switched by a logic circuit which controlled its activation and deactivation based on the logic output level of a counter. The counter was used to count movements. Its output level changed after four counts. Thus, when the velocity display was being controlled by the counter, visual feedback (knowledge of results) of peak flexion velocity was alternately supplied to the subject for four movements, then withheld from the subject for four movements.

To minimize memory errors when matching velocities after knowledge of results had been withdrawn, the number of movements per block was kept to a minimum. Four movements was felt to be the minimum number which would still give the subject enough of an opportunity to adjust to the altered visual feedback conditions.

The position display was regulated by a logic circuit that switched two LED's to signal an angular position target corresponding to the angle of extension from which flexion was to be initiated. The circuit consisted of two comparators wired to two LED's mounted one above the other. The comparators were wired in such a way that when the input voltage signal was between their thresholds both LED's lit up. When the input voltage was below the lower threshold only the bottom LED lit up and when it was above the upper threshold only the top LED lit up. Thus, when the angle of extension was within the limits of the target window both LED's would light up informing the subject that the thumb

was properly positioned to initiate a 'ballistic' flexion movement. The angular position window was approximately 0.13 rad wide.

The relative position of the target window could be changed without altering its width by means of a relay which added or subtracted resistance from the display circuit. The display circuit could also be switched by the counting circuit so that the initial angle would change in conjuction with activation or deactivation of the velocity display every four movements. The position display always remained active. A change in the output level of the counter only affected the angular position of the target window.

To facilitate data analysis, rectified EPL and FPL e.m.g.'s, angular position and angular velocity were averaged on-line. All four signals were filtered and amplified as described in the preceding chapter, before being digitized. The analog signals were sampled at 2 kHz. The averaging routine was capable of saving data acquired up to 500 ms prior to a trigger pulse. This feature allowed the averaging sweep to be triggered from the velocity signal without losing information about the portion of the FPL e.m.g. burst which preceded movement onset.

The trigger pulse was generated by a Schmitt trigger, triggering from the angular velocity signal. The trigger threshold was adjusted to a level slightly above that of the small velocity fluctuations which occurred during thumb positioning prior to movement initiation. The averaging

sweep, therefore, began shortly after movement onset, but included sampled data acquired before this time, as described above.

Subjects were told that the velocity display measured peak flexion velocity. They were instructed to aim for a target velocity which was indicated by two LED's mounted side by side in the sixth position of the LED column. Both LED's flashed when peak flexion velocity fell within the target window. When an LED above the target flashed, the subject knew that the peak flexion velocity achieved during that movement had fallen short of the target. Similarly, when an LED below the target flashed the subject knew that the target velocity had been overshot.

Subjects were allowed to practice until satisfied with the degree of consistency they had achieved from one movement to the next. Since it was not possible to be on-target with 100% reliability, even after considerable practice, no attempt was made to impose a criterion for consistency. During averaging, no discrimination was made between successful and unsuccessful attempts to achieve the target velocity. Each trial consisted of 40 movements which were executed without interruption unless technical problems arose or the subject expressed dissatisfaction with the performance. If a trial had to be interrupted it was repeated in its entirety.

During practice, subjects were provided with a knowledge of results (in the form of the LED velocity

display) for each movement. During trials, the velocity display was switched by the counting circuit. In some experiments the position display was also switched by the counting circuit. Thus, the state of the velocity display (and in some experiments also the state of the angular position display) changed predictably after each set of four movements during a trial. Movements recorded when there was knowledge of results and when there was no knowledge of results were averaged in separate arrays. Each trial, therefore, resulted in two 20 movement averages, one of which represented a visual feedback condition (knowledge of results), the other a no-visual feedback condition (no knowledge of results).

During practice, subjects were always asked to produce maximal velocities under each of the experimental conditions. This ensured that any undershoot of the velocity target during an experiment was not due to the subject's physical limitations.

Experiment 1

Eight subjects participated in both Experiment 1 and Experiment 2. Experiment 1 established a baseline for the effect of withdrawing knowledge of results during the no-visual feedback condition. It also examined the effect of changing the initial angle from which flexion was initiated, which also altered the movement amplitude.

Subjects were instructed to regulate flexion so as to match peak flexion velocity under both the visual feedback and no-visual feedback conditions. Two sets of three pairs of trials were run. In the first trial of each pair, the initial angle remained constant. In the second trial of the pair the initial angle changed according to the state of the velocity display (visual feedback or no-visual feedback). When there was visual feedback the initial angle was the same as it had been during the first trial of the pair. When there was no visual feedback the initial angle was reduced in trials belonging to the first set and was increased in trials belonging to the second set.

Each pair of trials in a set had a different target velocity which was changed by adjusting the velocity gain. In the first set the target velocities were 6.7, 10.2 and 13.6 rad/s. In the second set they were 9.0, 13.6 and 18.1 rad/s. The initial angle from which flexion was to be initiated was approximately 0.55 rad in the visual feedback condition, 0.35 rad in the no-visual feedback condition for the first set of trials and 0.75 rad for the second set. Each set of trials was carried out in increasing order of target velocity.

Experiment 2

Experiment 2 attempted to establish the sensitivity of the subjects' judgment of flexion velocity during voluntary movements. Three pairs of trials were run, each pair having

a different target velocity. The target velocities were 9.0, 13.6 and 18.1 rad/s. Trials were carried out in increasing order of target velocity. All movements began from an angular position of approximately 0.55 rad. In the first trial of each pair the subject aimed for a target velocity during the visual feedback condition, as in the previous experiment. However, during the no-visual feedback condition, the subject was instructed to make movements which were, according to the subject's judgment, slightly faster than the movements under the visual feedback condition. The same procedure was used in the second trial of the pair except that the subject was instructed to make slower rather than faster movements. It was impressed upon the subject that these movements be only as much faster or slower than the movements made when visual feedback was available, as was necessary for them to be perceived as being faster or slower.

Experiment 3

Experiment 3 and Experiment 4 were carried out to determine whether the effects seen in Experiment 1 were due to the change in movement amplitude or the change in the initial angle from which flexion was initiated.

To facilitate these experiments, the relay in the circuit controlling the position display was also wired into a second circuit which regulated the switching of a retractable stop. The retractable stop consisted of a

solenoid which was securely clamped to a rigid support. When activated, the core-element was pulled forward creating a mechanical stop about 0.2 rad above another fixed stop. When deactivated, the core-element was pulled back by a spring, retracting the upper stop.

Six subjects participated in Experiment 3. All six had also participated in Experiments 1 and 2. In Experiment 3 the initial angle remained the same under both visual feedback and no-visual feedback conditions, but the retractable stop was interposed under the visual feedback condition only. Thus, although the initial angle was the same, the movement amplitude was greater under the no-visual feedback than under the visual feedback condition.

Subjects were instructed, as before, to regulate flexion so as to match the target velocity under both visual feedback and no-visual feedback conditions. Three trials were run using target velocities of 9.0, 13.6 and 18.1 rad/s respectively. Trials were carried out in increasing order of target velocity. Flexion was initiated from an angle of approximately 0.75 rad. Under the visual feedback condition movement amplitude was, therefore, limited to approximately 0.55 rad, while it was increased to approximately 0.75 rad under the no-visual feedback condition.

Experiment 4

Six subjects participated in Experiment 4. Of these, five had also participated in Experiments 1, 2 and 3. In

Experiment 4 movement amplitude was kept constant while the initial angle alternated. Under the visual feedback condition the retractable stop was withdrawn and the position display signaled an initial angle of approximately 0.55 rad. Under the no-visual feedback condition the stop was in place and the position display signaled an initial angle of approximately 0.75 rad. Thus, in both conditions the movement amplitude was held at approximately 0.55 rad. Again, there were three trials with target velocities of 9.0, 13.6 and 18.1 rad/s respectively and trials were carried out in increasing order of target velocity.

Experiment 5

Experiment 5 was carried out to determine whether judgment of velocity was dependent on the force opposing motion. Force was applied to the rotational axis of the apparatus in Figure 4 by coupling it to a printed circuit motor. The circuit controlling the motor employed a negative velocity-feedback loop to produce a force opposing motion which was similar to viscous friction. The velocity-feedback loop was wired in series with a relay which permitted the loop to be interrupted either when the velocity display was activated or when it was deactivated. When the velocity-feedback loop was interrupted there was a residual load consisting of the inertia of the printed circuit element and the friction of the brushes contacting it. The position display was disconnected from the counting circuit

so that the initial angle would be the same under visual feedback and no-visual feedback conditions.

Six subjects participated in Experiment 5. All six had participated in Experiments 1, 2 and 3. Five had also participated in Experiment 4. As in other experiments, they were instructed to move so as to match the target velocity under both visual feedback and no-visual feedback conditions. Two sets of three trials were run. In the first set the viscous load was introduced only under the no-visual feedback condition. In the second set the situation was reversed with the viscous load present only under the visual feedback condition. The three target velocities were 7.8, 10.9 and 13.6 rad/s. Trials were carried out in increasing order of target velocity.

Subjects were allowed to practice a few movements with the load before beginning the first set of trials, but were not given knowledge of results.

Experiment 6

Experiment 6 was carried out to examine how the length-tension characteristics of the EPL and FPL muscles affected the peak angular velocity of flexion. The author was the only subject of this experiment.

A mechanical stop was set to limit extension. There was no stop to restrict flexion. The thumb was positioned against the stop and held there either voluntarily or by the action of a weak spring. In the first case a small amount of

activity was required in EPL; in the second case EPL was relaxed. The median nerve of the right arm was stimulated near the elbow using the twin-pulse output of a Grass S9 stimulator.

There were two protocols. In the first, the strength of the stimulus was set above the level which produced a maximal e.m.g. response in the FPL muscle. By carefully probing the nerve it was possible to find a position where the stimulus was quite selective for FPL. Five stop positions were chosen and at each position the responses to ten stimuli were digitized and averaged. The sampling sweep was triggered from the stimulator.

In the second protocol, the stop was fixed in one position and five different stimulus levels were chosen. Ten responses to each stimulus level were digitized and averaged.

B. Results

Experiment 1

Figure 15 illustrates the effect of the withdrawal of knowledge of results on the ability to match a given target velocity. There is very little difference between movements made under visual feedback and no-visual feedback conditions. Table 2 gives the mean change in peak flexion velocity and movement time under the two conditions for the eight subjects. Neither of these means were significantly

ANGULAR VELOCITY

5 rad/s

ANGULAR POSITION

0.2 rad

FPL EMG

EPL EMG

100 ms
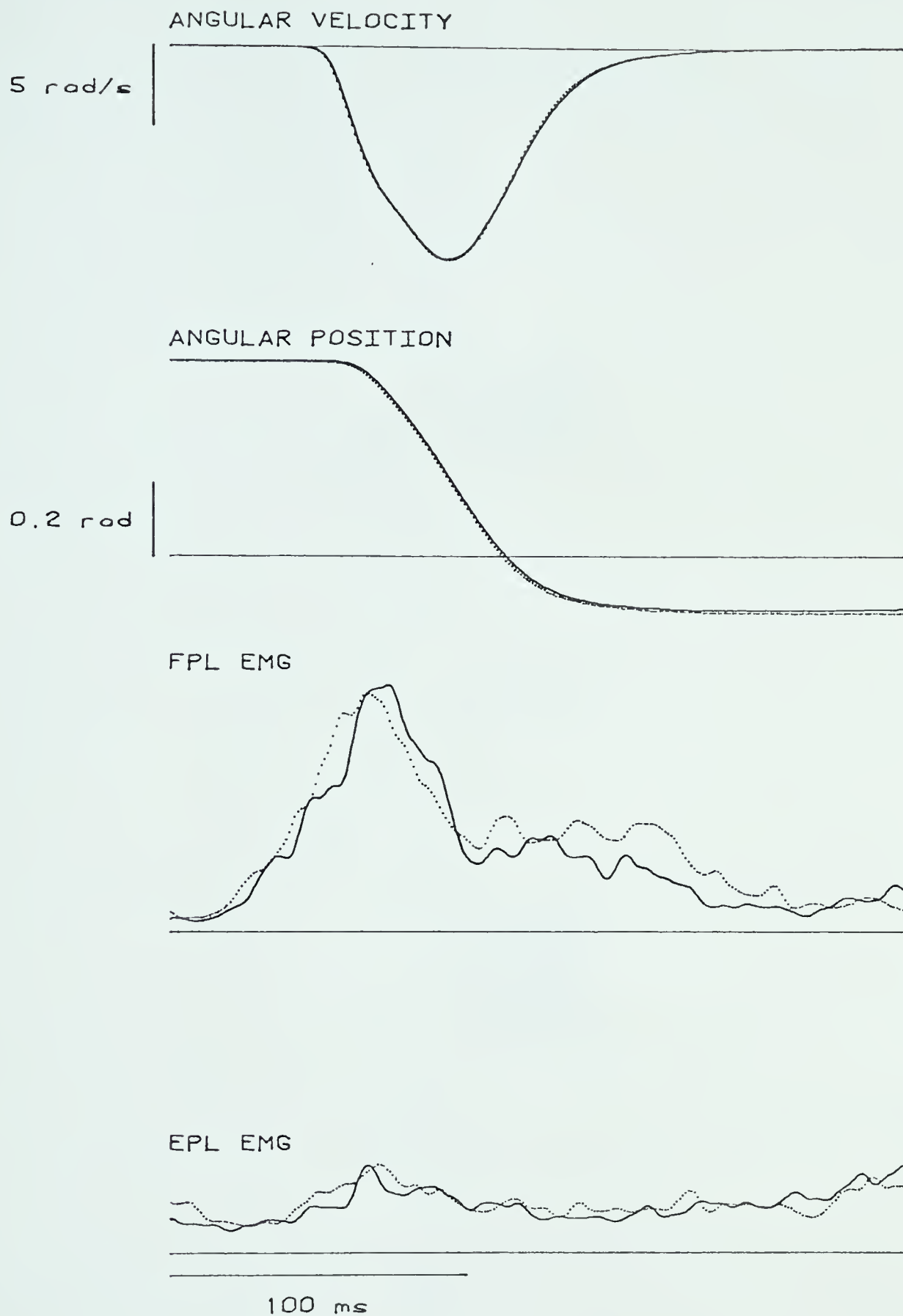
FIGURE 15. Solid lines and dotted lines represent averages of 20 movements made respectively with and without visual feedback of peak angular velocity. Angular position and velocity trajectories are almost identical and e.m.g. profiles are very similar when subjects attempt to match peak velocity under the two visual feedback conditions.

TABLE 2

Changes in Peak Velocity and Movement Time: Variable=Feedback

Target Velocity(rad/s)	Mean Change in Velocity(rad/s)[s.d.]	n	Mean Change in Movement Time(ms)[s.d.]	n
6.7	0.10[0.40]	8	7.0[11.2]	8
10.2	0.25[0.54]	8	-2.2[5.3]	7
13.6	0.20[0.57]	8	-1.6[5.1]	8
9.0	0.32[0.51]	8	0.2[5.4]	8
13.6	-0.12[0.45]	7	1.6[3.6]	7
18.1	0.09[1.04]	8	1.6[6.1]	8

different from zero for any given target velocity (2P>0.3 for velocity; 2P>0.1 for movement time). Statistical analysis of differences between population means involved a two-sample t-test (when the test is two-tailed the notation 2P is used; when it is one-tailed the notation P is used).

Figures 16 and 17 illustrate the effects of changing both the initial angle and movement amplitude on the ability to match velocities under visual feedback and no-visual feedback conditions. When the initial angle was reduced the peak flexion velocity was consistently lower during the no-visual feedback than the visual feedback condition (Figure 16). When the initial angle was increased the velocity was consistently higher (Figure 17). The velocity undershoot was usually accompanied by a reduction in the size of the integrated FPL e.m.g. burst while the overshoot was accompanied by an increment, although these effects occurred even when the e.m.g. changes were minimal.

Table 3 gives the mean undershoot or overshoot in velocity for the subjects at each target velocity, as well as the mean change in movement time. The undershoots were significantly greater than zero for target velocities of 10.2 and 13.6 rad/s (P<0.0005), but not for 6.7 rad/s (0.05<P<0.1). The overshoots were significantly greater than zero for all three target velocities (P<0.01). In the five cases where undershoots or overshoots were significantly greater than zero they were also significantly greater than the corresponding mean change in peak flexion velocity

FIGURE 16. Reducing initial angle and movement amplitude when visual feedback of peak velocity was withheld (dotted lines) caused the subject to undershoot peak velocity when attempting to match the peak velocity of the visual feedback condition (solid lines) even though FPL e.m.g.'s were often very similar.

ANGULAR VELOCITY

5 rad/s
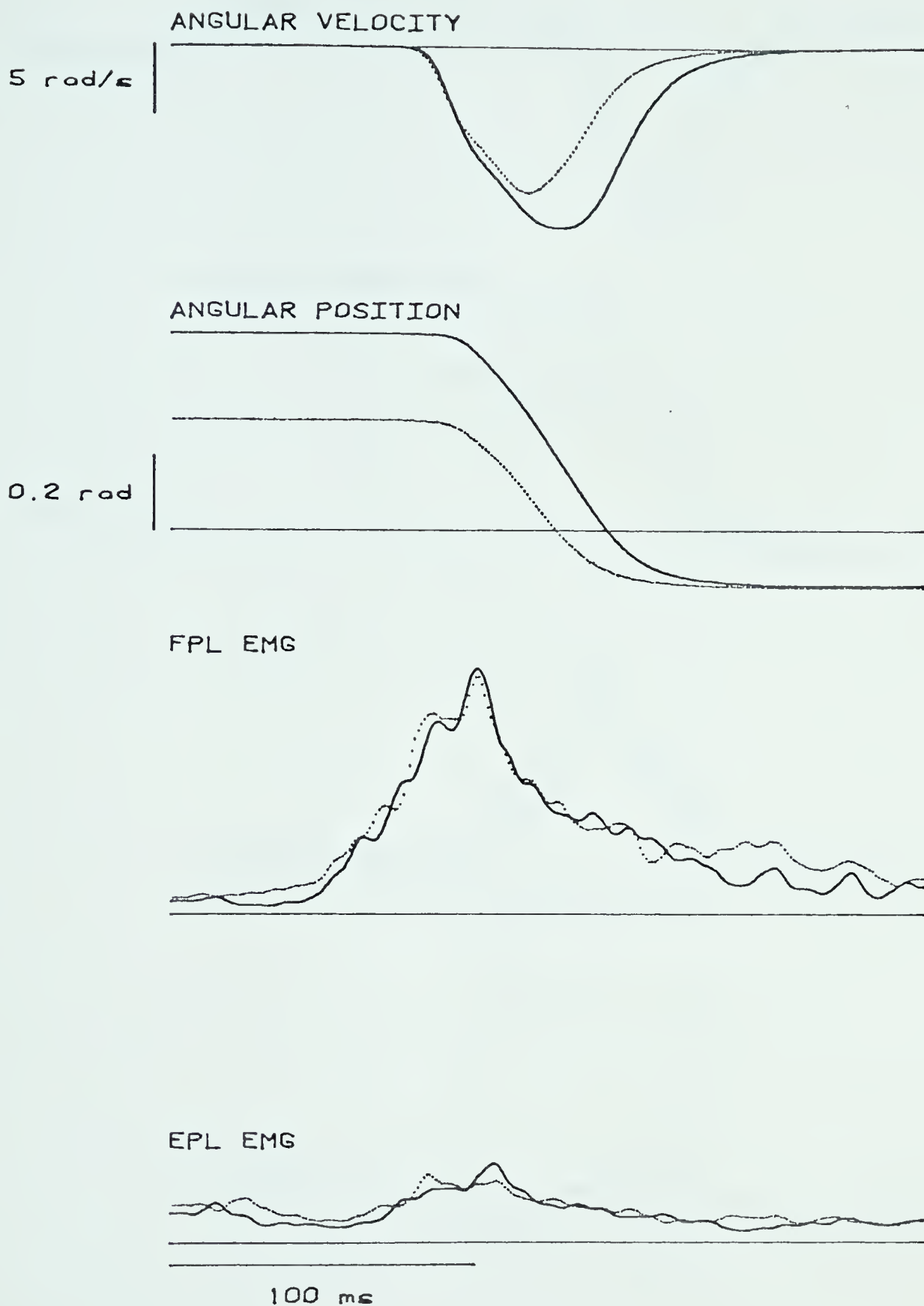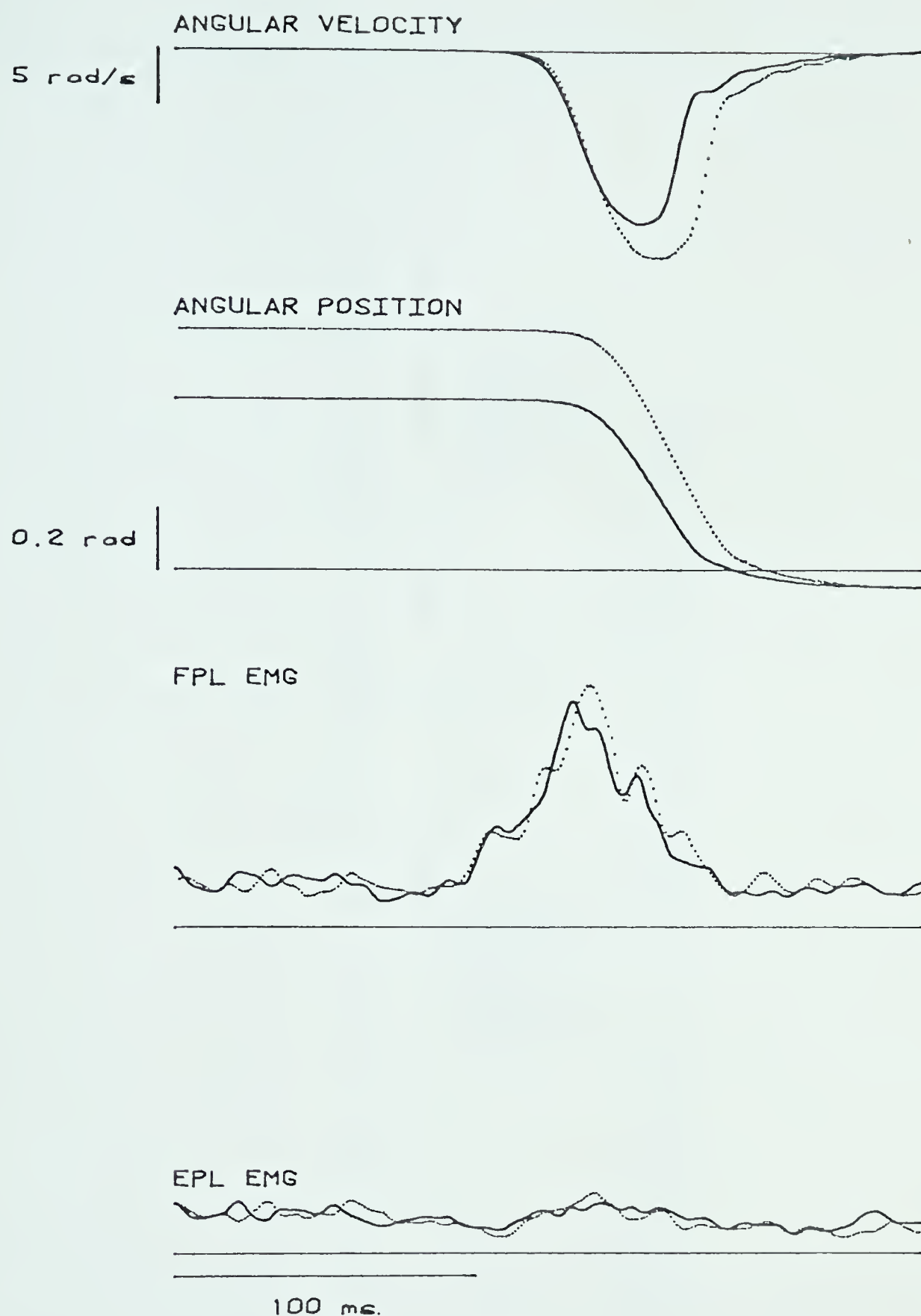
ANGULAR POSITION

0.2 rad

FPL EMG

EPL EMG

100 ms.

FIGURE 17. Increasing initial angle and movement amplitude when visual feedback of peak velocity was withheld (dotted lines) caused the subject to overshoot peak velocity when attempting to match the peak velocity of the visual feedback condition (solid lines) even though FPL e.m.g.'s were often very similar.

TABLE 3

Changes in Peak Velocity and Movement Time: Variables=Feedback, Amplitude and Initial Angle

Target Velocity(rad/s)	Mean Change in Velocity(rad/s)[s.d.]	n	Mean Change in Movement Time(ms)[s.d.]	n
6.7	-0.47[0.93]*	8	-31.3[17.7]*	8
10.2	-1.42[0.78]*	8	-26.9[9.9]*	7
13.6	-2.89[0.84]*	8	-15.7[4.8]*	8
9.0	1.66[1.24]**	8	24.7[8.0]**	8
13.6	1.74[1.46]**	7	15.1[10.2]**	7
18.1	2.66[1.26]**	8	10.7[5.4]**	8

*Decrement of 0.2 rad in amplitude and initial angle during no-visual feedback condition
**Increment of 0.2 rad in amplitude and initial angle during no-visual feedback condition

obtained when the initial angle remained constant under both visual feedback and no-visual feedback conditions (P<0.01).

Whenever the initial angle was reduced during the no-visual feedback condition there was a decrement in movement time which was significantly greater than zero (P<0.0025) and significantly greater than the change in movement time obtained when the initial angle did not differ between visual feedback and no-visual feedback conditions (P<0.005). Correspondingly, whenever the initial angle was increased there was an increment in movement time which was significantly greater than zero (P<0.005) and significantly greater than the change in movement time when the initial angle remained fixed (P<0.005).

In Figures 16 and 17 the velocity trajectories of movements beginning from different positions are remarkably similar for about the first 30 ms when the subject is attempting to match peak velocities. For comparison Figure 18 shows the velocity trajectories of the same subjects for a series of different target velocities all initiated from the same position.

Experiment 2

Table 4 summarizes the results of instructing subjects to make movements during the no-visual feedback condition which were just perceptibly slower or faster than those made under the visual feedback condition. The mean decrement or increment with respect to the target velocity was always
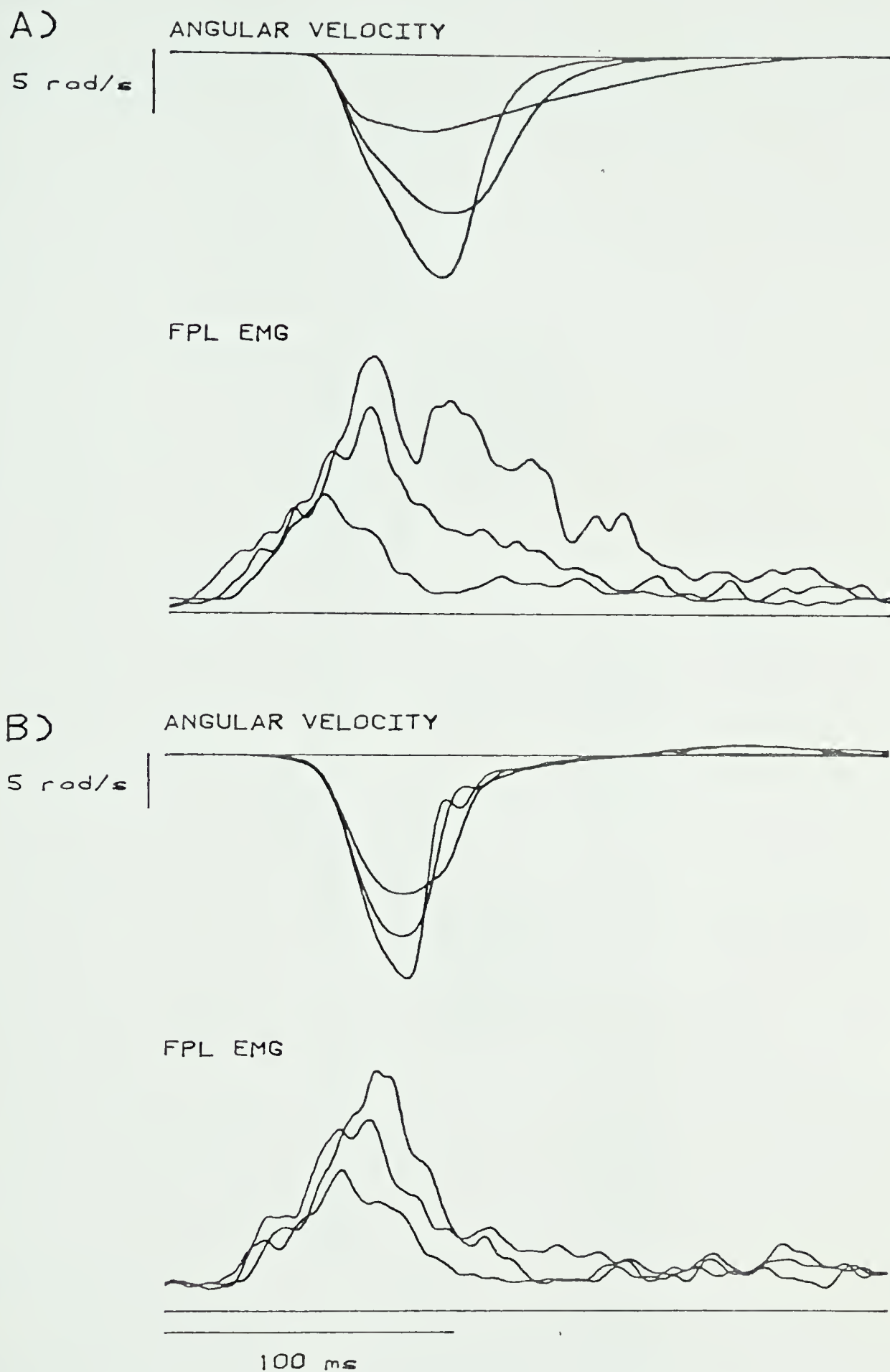
FIGURE 18. Velocity trajectories and FPL e.m.g. profiles for movements with three different target velocities. All three movements were initiated from the same angle. The initial phases of the velocity trajectories were very similar. A) Subject of Figure 16. B) Subject of Figure 17.

TABLE 4

Changes in Peak Velocity and Movement Time: Variables=Feedback and Velocity

Target Velocity(rad/s)	Mean Change in Velocity(rad/s)[s.d.]	n	Mean Change in Movement Time(ms)[s.d.]	n
9.0	-1.68[0.80]*	7	23.3[23.7]*	7
13.6	-2.95[1.57]*	7	18.1[11.8]*	7
18.1	-3.08[1.04]*	6	13.4[8.0]*	6
9.0	2.14[1.49]**	7	-18.1[11.8]**	7
13.6	2.85[1.92]**	7	-5.6[7.3]**	7
18.1	2.61[0.97]**	7	-5.6[5.8]**	7

*Slower movements were requested during the no-visual feedback condition
**Faster movements were requested during the no-visual feedback condition

significantly greater than zero (P<0.025). Movement times showed a significant increase for slower movements (P<0.025) and a significant decrease for faster movements (P<0.05). However, caution must be exercised in comparing Tables 3 and 4 since initial angle changed in the former, but not in the latter case. For similar target velocities, the velocity undershoots and overshoots were within the range of the corresponding velocity decrements and increments that could be judged by the subjects.

Since the initial angle and movement amplitude were not varied in Experiment 2, linear regression analysis was carried out to determine with which of several FPL e.m.g. parameters, the peak flexion velocity was best correlated. The parameters tested were e.m.g. burst amplitude, duration, integrated area and slope of the rising phase of the burst. The poorest correlation was obtained between velocity and burst duration (Brown and Cooke, 1981). The correlation coefficients were less than 0.44 for five of the seven subjects tested. The other three parameters correlated almost equally well with velocity (Figure 19). In nearly all cases the correlation coefficients were greater than 0.9.

Experiment 3

Figure 20 illustrates the effects of increasing movement amplitude without changing the initial angle. Peak flexion velocity was essentially the same although the movement lasted substantially longer. This longer movement
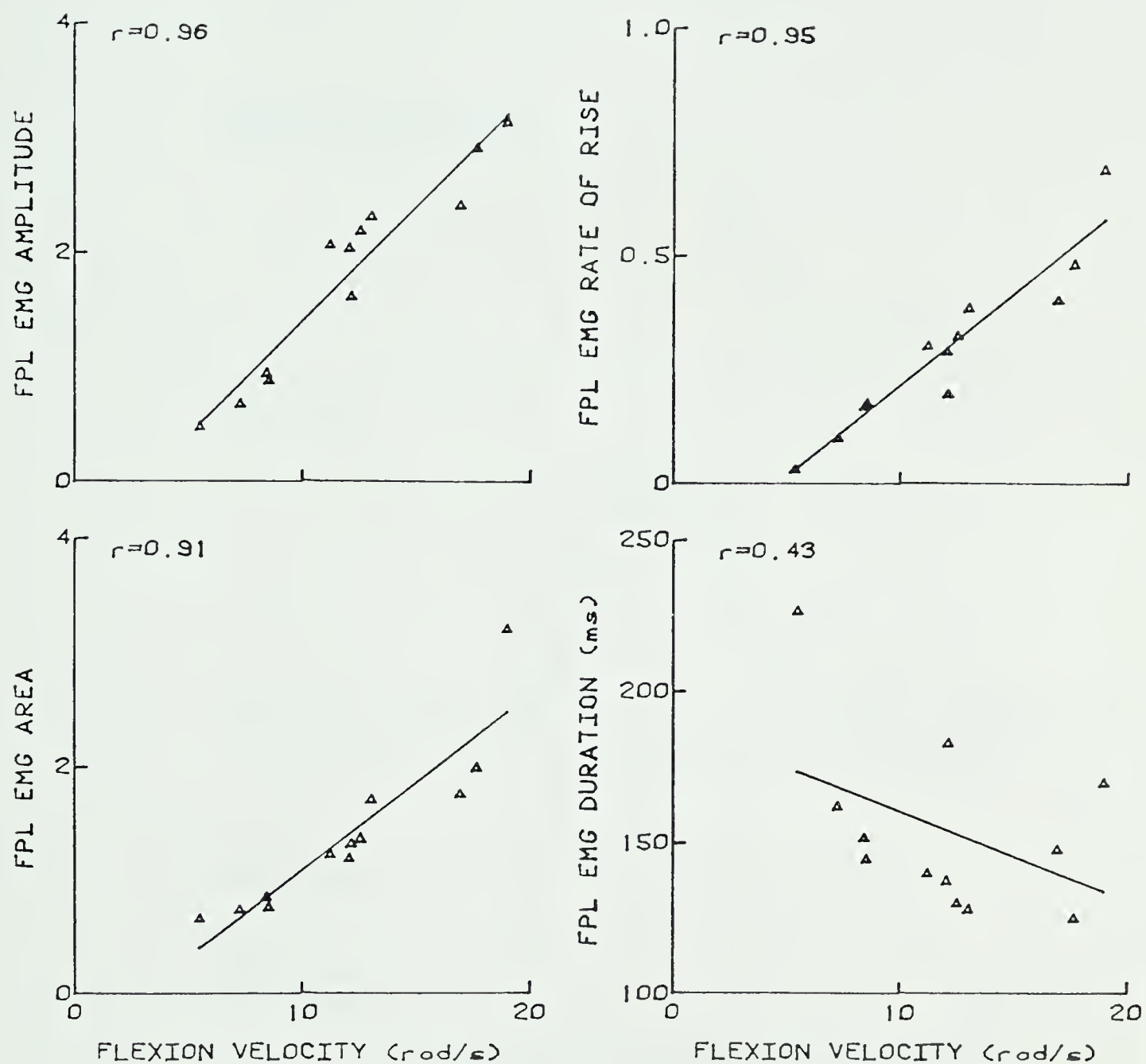
FIGURE 19. Plots of FPL e.m.g. amplitude, integrated area, rate of rise and duration vs peak flexion velocity. All plots are for the same subject.
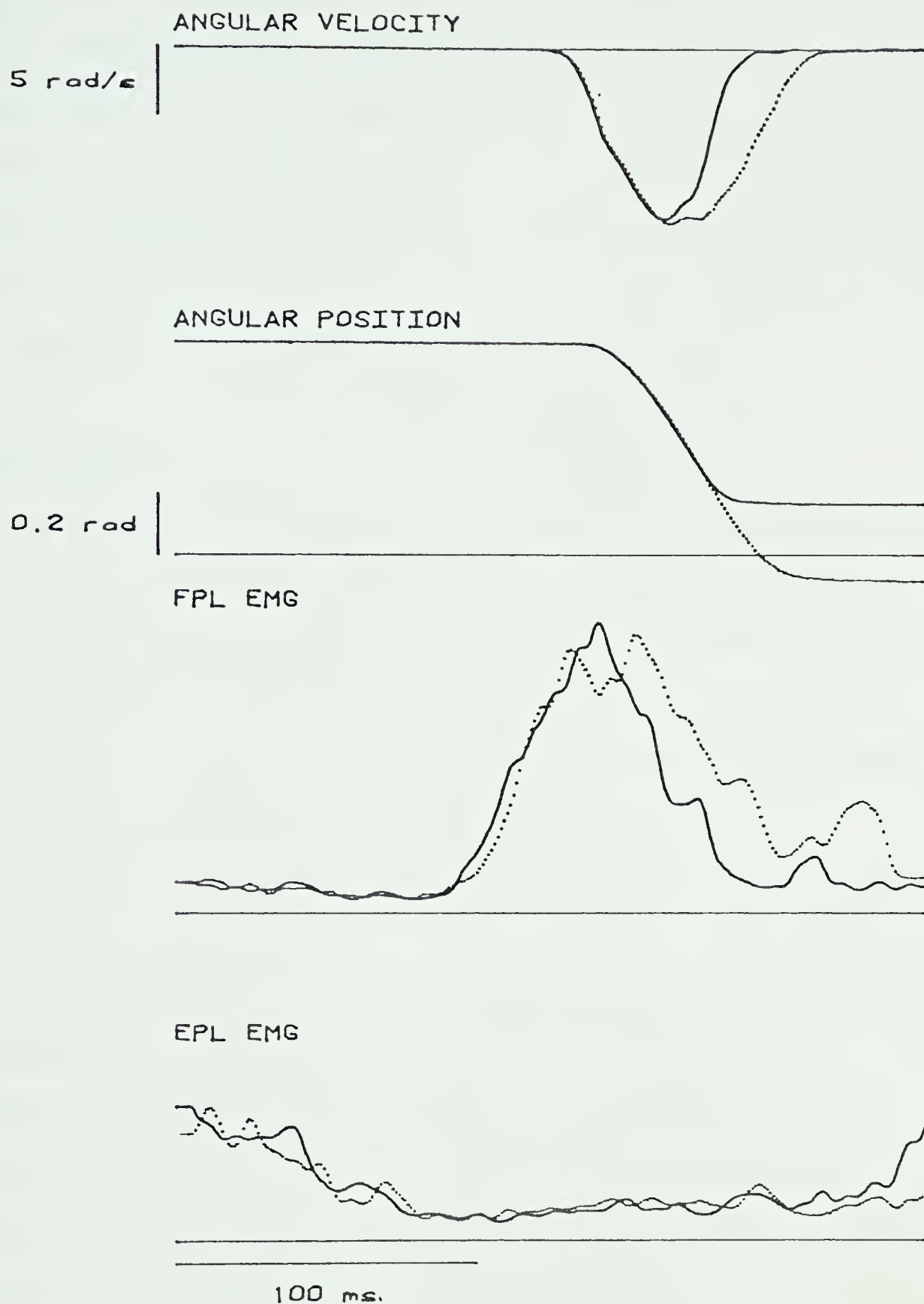
FIGURE 20. Increasing movement amplitude without changing initial angle when visual feedback of peak velocity was withheld (dotted lines) produced little difference in peak velocity when attempting to match the peak velocity of the visual feedback condition (solid lines). FPL e.m.g.'s were often increased in duration.

duration is reflected in an increased duration of the FPL burst (Wadman et al., 1979; Mishima et al., 1981). Table 5 shows that while the mean change in velocity for the six subjects is not significantly different from zero under visual feedback and no-visual feedback conditions for any target velocity (2P>0.6), the mean change in movement time is significantly greater than zero (P<0.0025).

Experiment 4

Figure 21 illustrates the effect of increasing the initial angle without changing the movement amplitude. The velocity overshoot under the no-visual feedback condition was sometimes accompanied by a larger FPL e.m.g. burst, but occurred even when the e.m.g. burst showed little change from that of the visual feedback condition.

Table 6 gives the mean overshoot for the six subjects for each target velocity. All are significantly greater than zero (P<0.01). They were also significantly greater than the corresponding differences observed in Experiment 3 (P<0.025). Table 6 also indicates, as expected, that velocity overshoots were accompanied by significant reductions in movement times under the no-visual feedback condition of Experiment 4 (P<0.05).

Experiment 5

The outcome of Experiment 5 was equivocal. Three subjects consistently produced a velocity undershoot when

TABLE 5

Changes in Peak Velocity and Movement Time: Variables=Feedback and Amplitude

Target Velocity(rad/s)	Mean Change in Velocity(rad/s)[s.d.]	n	Mean Change in Movement Time(ms)[s.d.]	n
9.0	-0.02[0.29]	6	40.7[6.6]	6
13.6	-0.21[0.97]	6	33.1[13.9]	6
18.1	-0.03[1.08]	6	24.5[7.7]	6
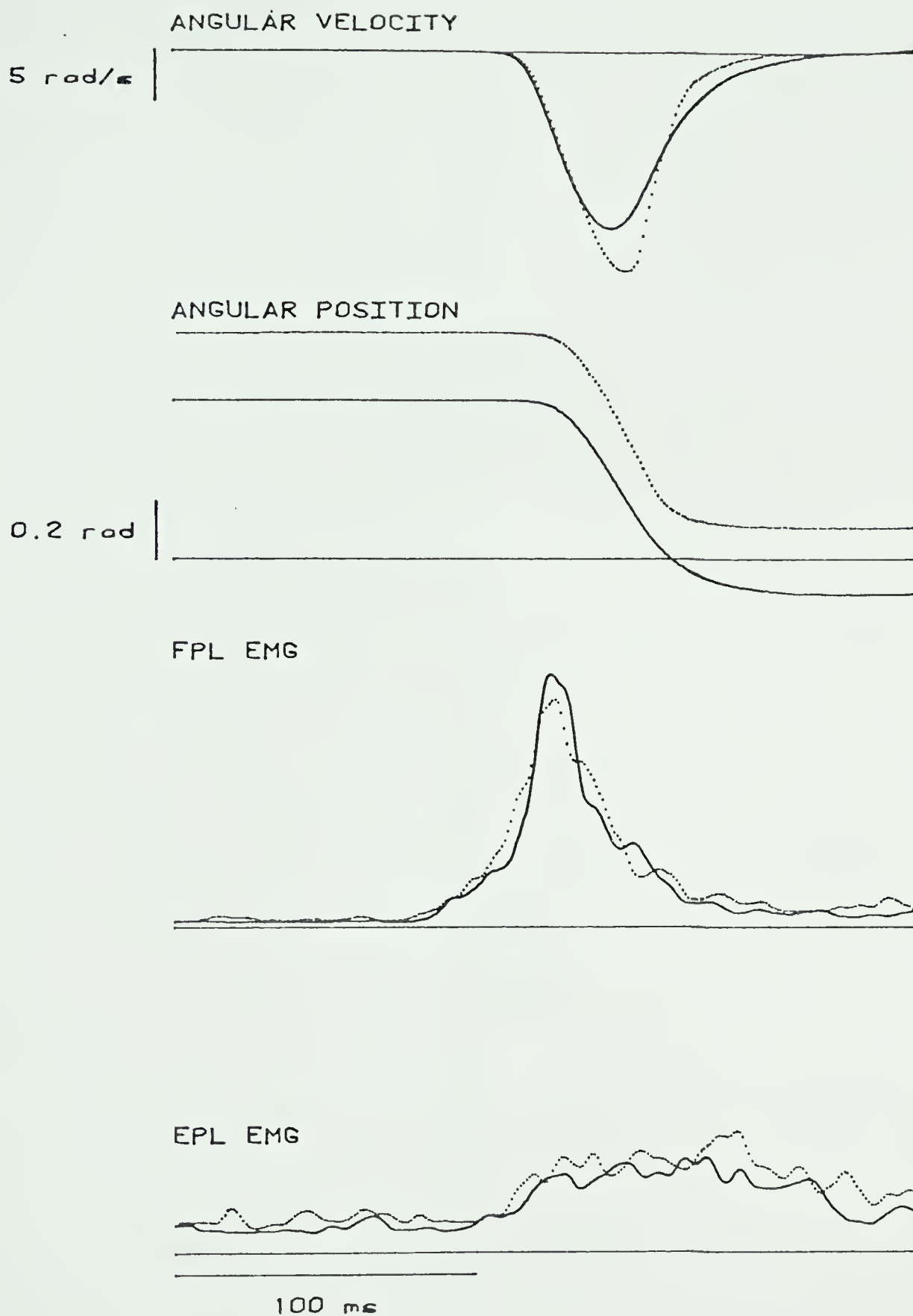
FIGURE 21. Increasing initial angle without increasing
movement amplitude when visual feedback of peak velocity was
withheld (dotted lines) caused subjects to overshoot peak
velocity when attempting to match the peak velocity of the
visual feedback condition (solid lines) even when FPL
e.m.g.'s were very similar.

TABLE 6

Changes in Peak Velocity and Movement Time: Variables=Feedback and Initial Angle

Target Velocity(rad/s)	Mean Change in Velocity(rad/s)[s.d.]	n	Mean Change in Movement Time(ms)[s.d.]	n
9.0	1.58[1.00]	6	-19.9[19.5]	6
13.6	1.74[0.93]	6	-7.4[7.5]	6
18.1	1.96[1.41]	6	-9.3[7.0]	6

the viscous load was introduced under the no-visual feedback condition and they consistently produced a velocity overshoot when the conditions were reversed. These undershoots (Figure 22) or overshoots (Figure 23) were often very large. The FPL e.m.g. records show that these errors resulted because there was little or no compensatory adjustment in the size of the burst.

Two subjects compensated much more adequately with appropriate increases (Figure 24) or decreases (Figure 25) in the size of the FPL e.m.g. burst. These adjustments were usually quite large, as shown.

The remaining subject produced undershoots similar to those of the first three subjects when the viscous load was introduced under no-visual feedback condition, but he also undershot when the conditions were reversed, as shown in Figure 26. There is a dramatic reduction in the amplitude of the corresponding FPL e.m.g. burst.

Experiment 6

Figures 27 and 28 show the responses obtained when the median nerve was stimulated supramaximally with the thumb positioned at various angles of extension. The results were similar whether the thumb was voluntarily extended or supported in extension by the action of a weak spring. The integrated FPL e.m.g. did not vary by more than 10% over the range of initial angles. This variation was not systematic, but was probably due to slight variations the position and
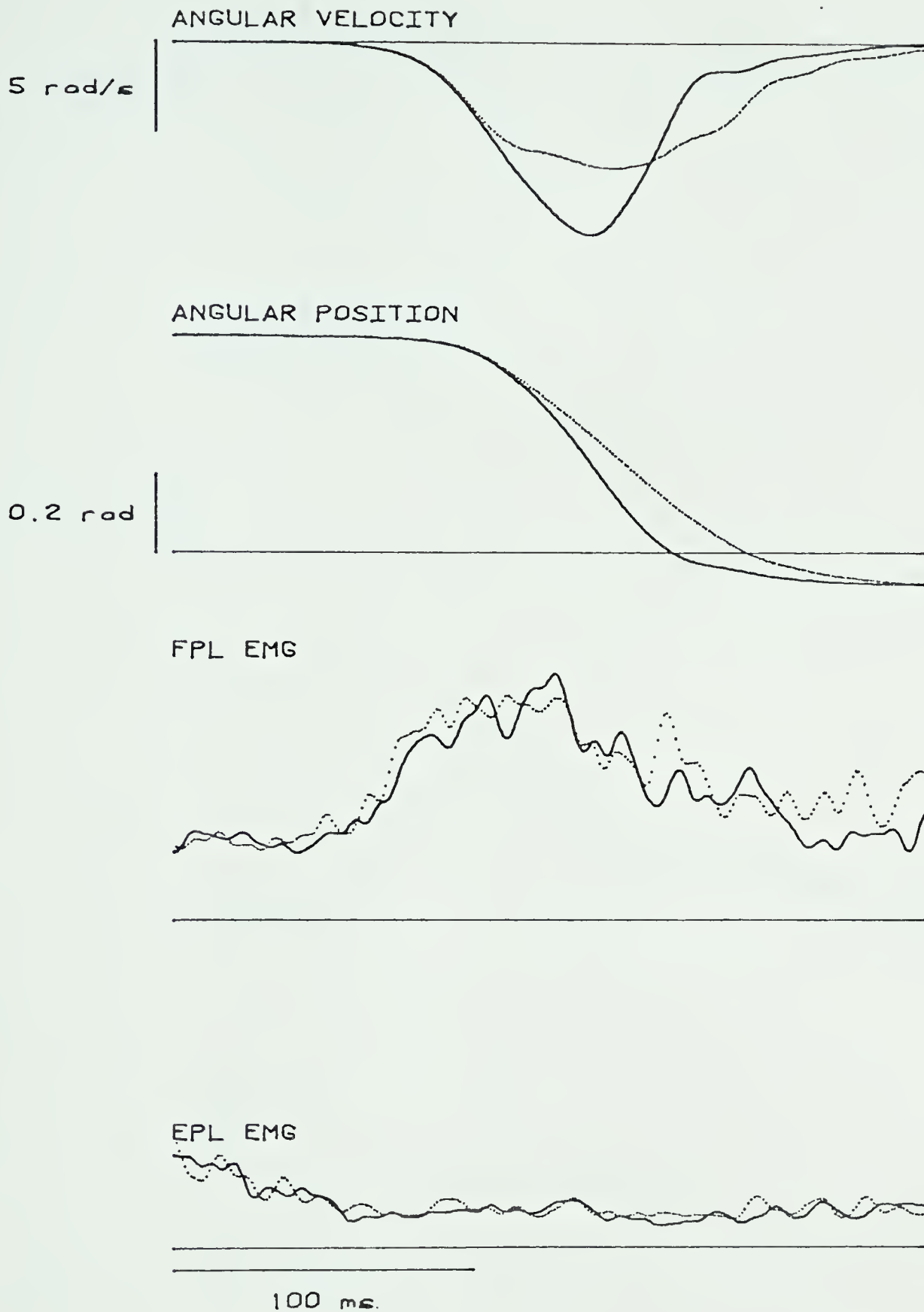
FIGURE 22. Introducing a viscous load when visual feedback of velocity was withheld (dotted lines) often caused subjects to undershoot peak velocity when attempting to match the peak velocity of the visual feedback condition (solid lines). FPL e.m.g.'s differed very little for the subject shown here.
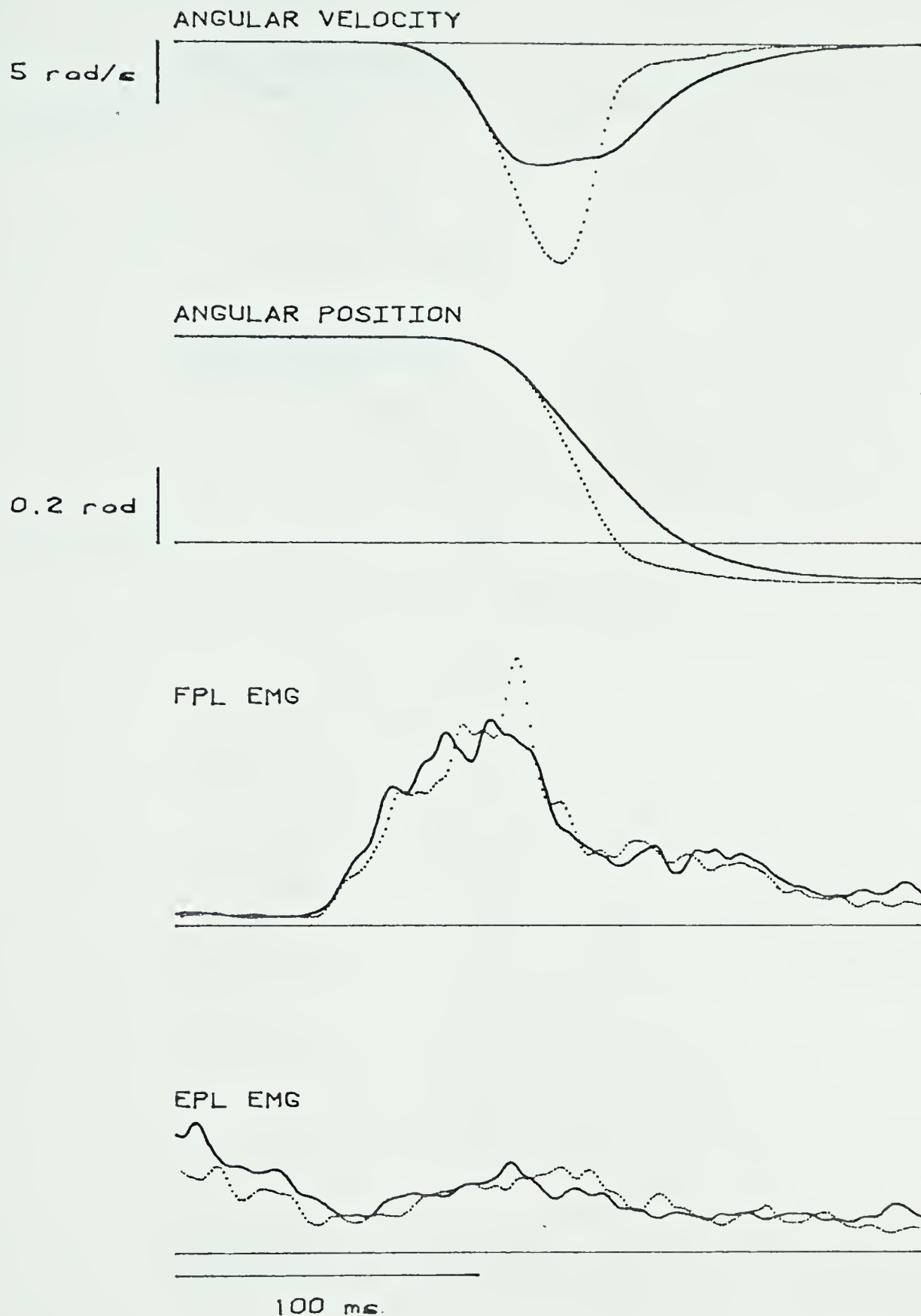
FIGURE 23. Removing a viscous load when visual feedback of velocity was withheld (dotted lines) often caused subjects to overshoot peak velocity when attempting to match the peak velocity of the visual feedback condition (solid lines). FPL e.m.g.'s differed very little for the subject shown here.

ANGULAR VELOCITY

5 rad/s

ANGULAR POSITION

0.2 rad

FPL EMG

EPL EMG

100 ms.
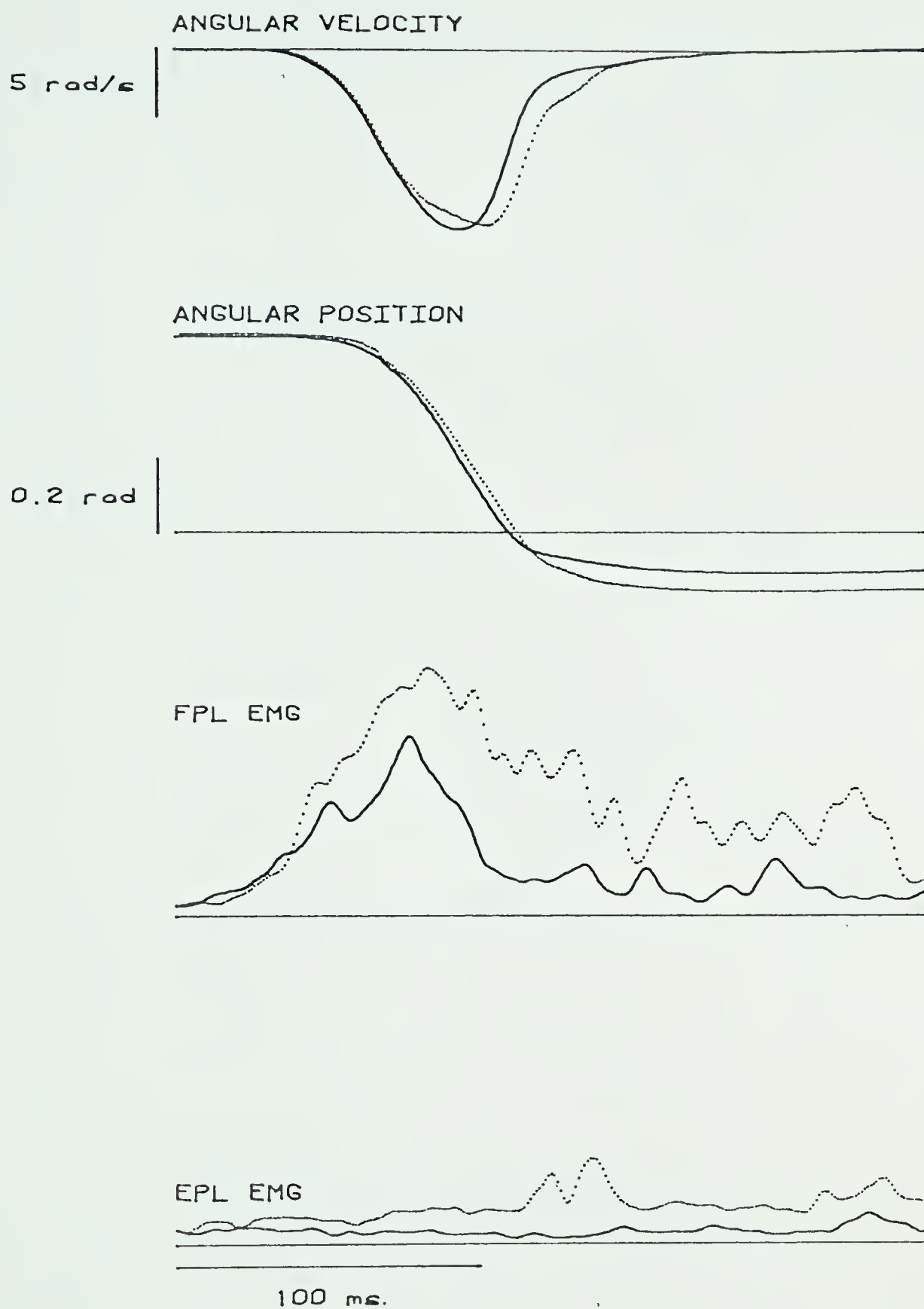
FIGURE 24. One of the two subjects who compensated adequately for introduction of the viscous load by increasing FPL activity when visual feedback of peak velocity was withheld (dotted lines).
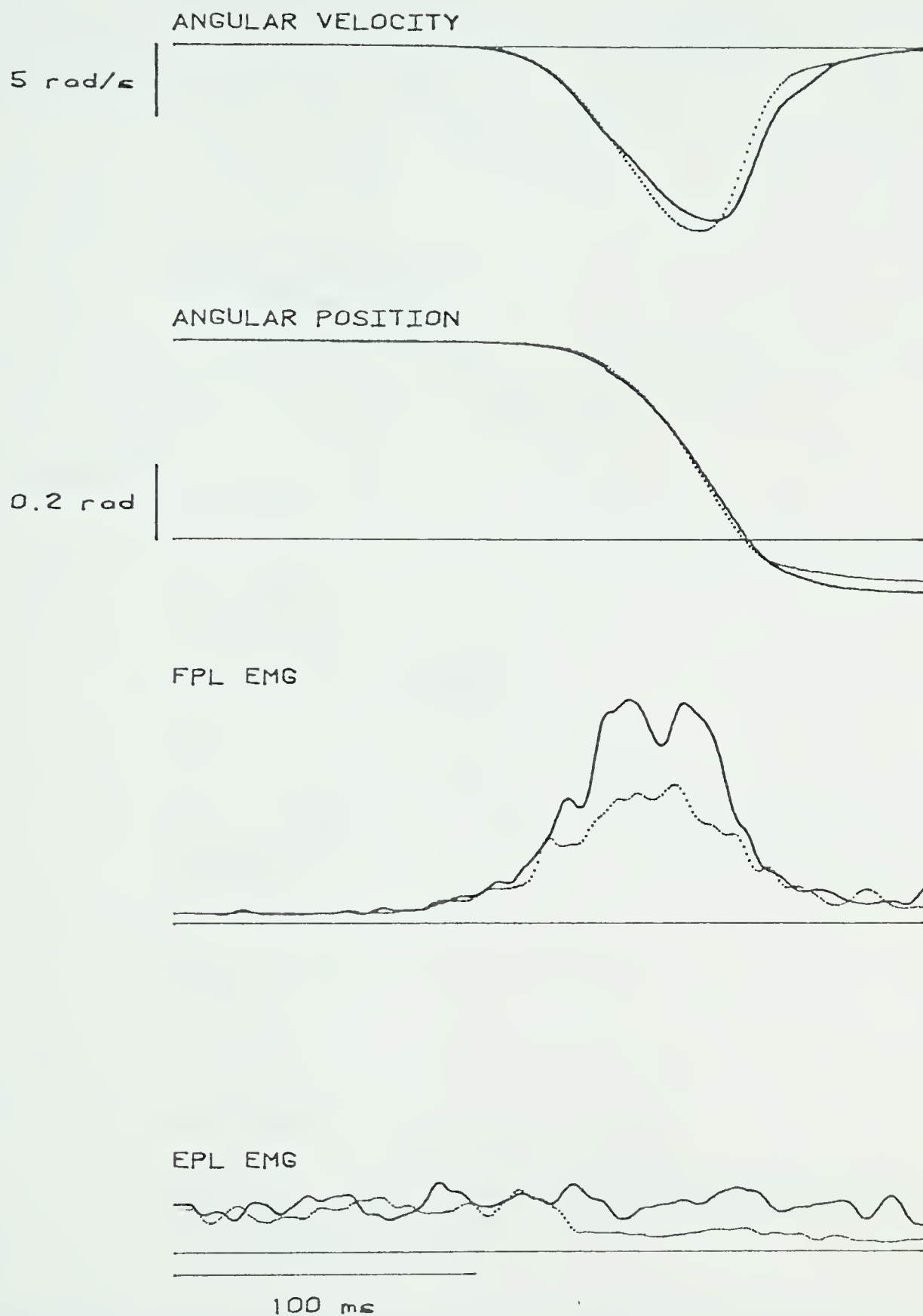
FIGURE 25. The same subject as in Figure 24 compensated
adequately for removal of the viscous load by reducing FPL
activity when visual feedback of peak velocity was withheld
(dotted lines).
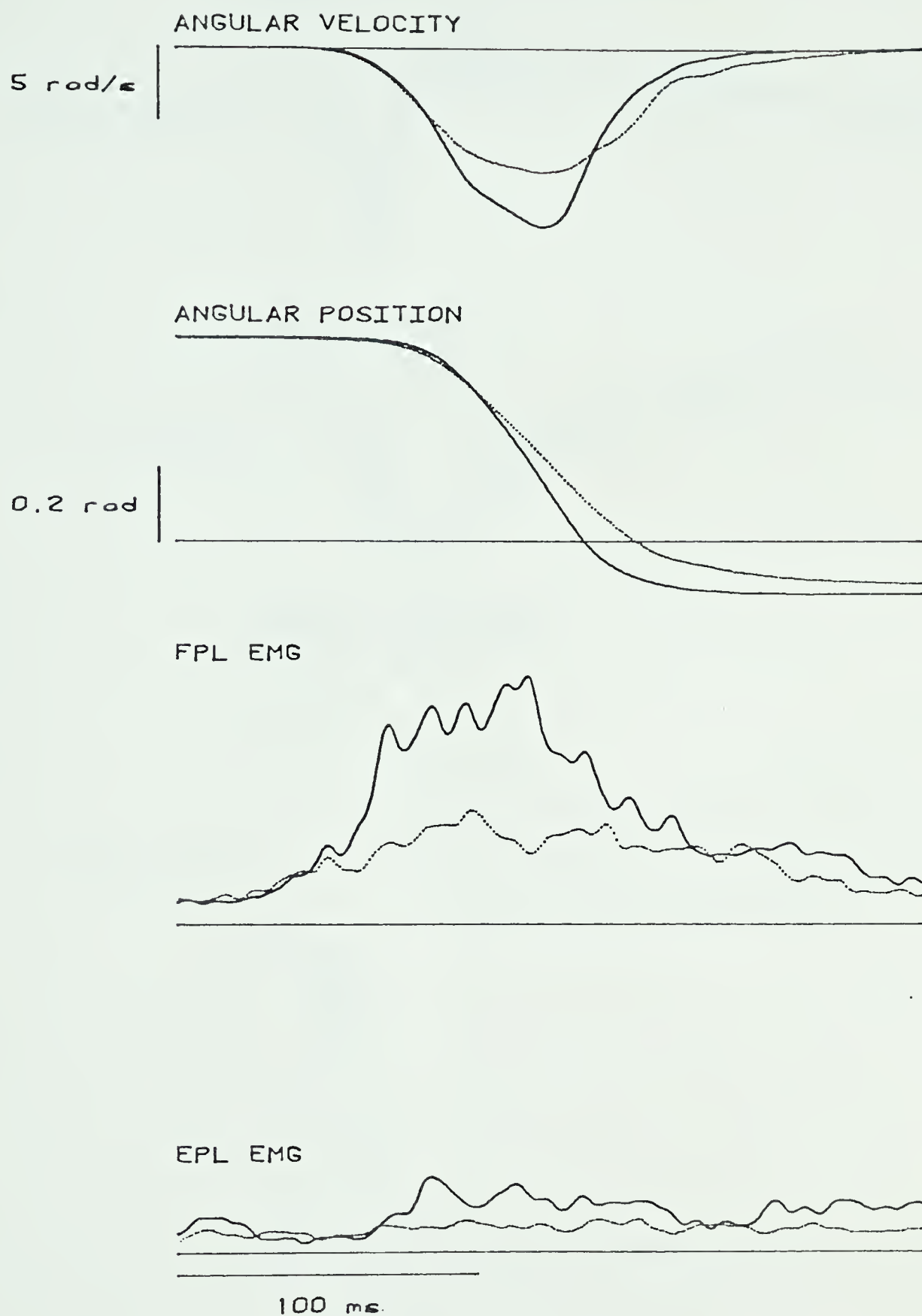
FIGURE 26. One subject overcompensated when the viscous load was removed and visual feedback of peak velocity was withheld (dotted lines). He reduced FPL activity much more than necessary causing an undershoot in peak velocity when attempting to match the velocity of the visual feedback condition (solid lines).

ANGULAR ACCELERATION

300 rad/s*s |

ANGULAR VELOCITY

5 rad/s |

ANGULAR POSITION

0.2 rad |

FPL EMG

100 ms
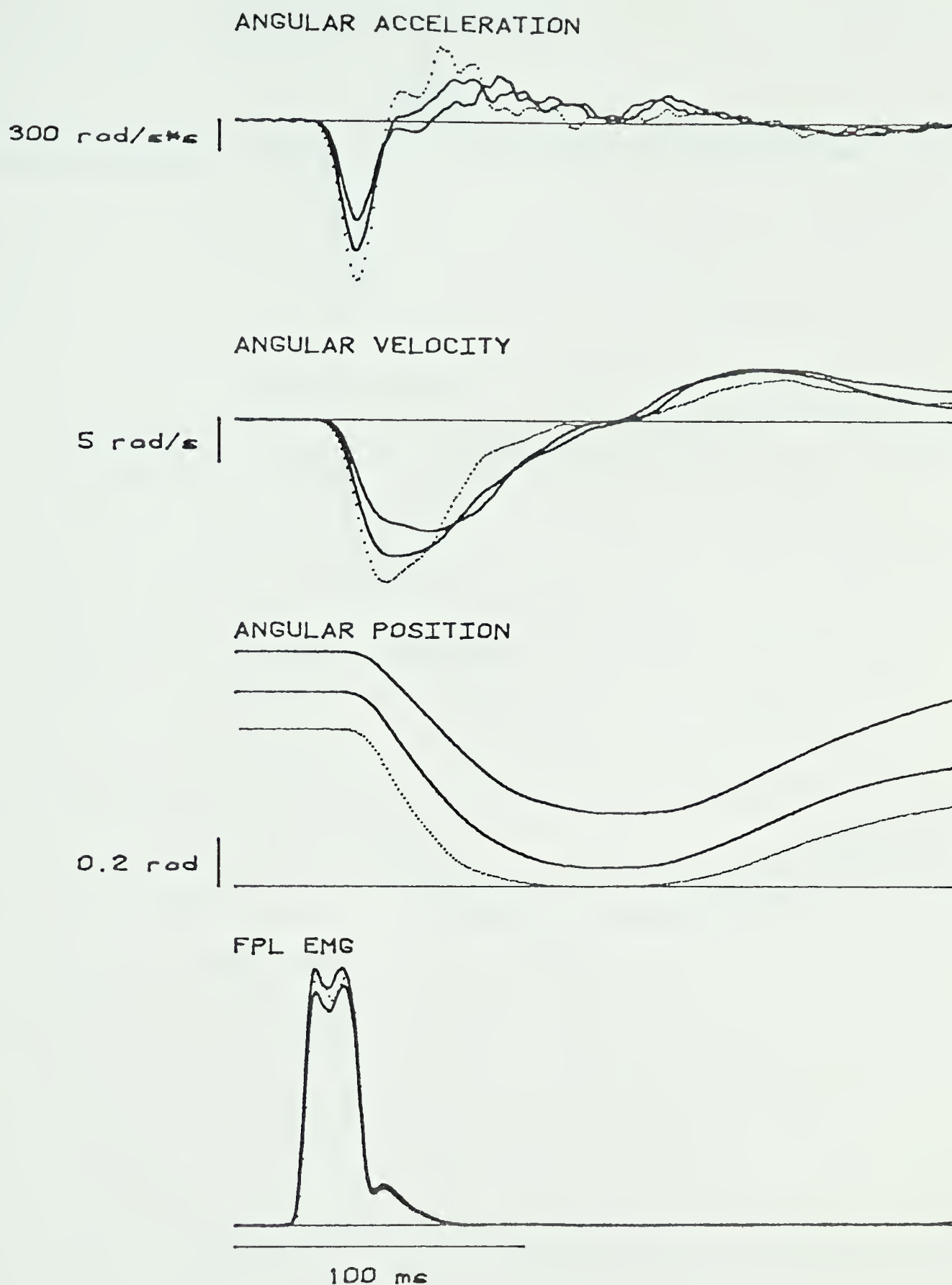
FIGURE 27. Stimulation of the median nerve to evoke a maximal e.m.g. response in the FPL muscle produced the greatest velocity and acceleration for an initial angle of approximately 0.6 rad (dotted lines). Peak velocity and acceleration delined progressively as the initial angle increased.

ANGULAR ACCELERATION

300 rad/s*s |

ANGULAR VELOCITY

5 rad/s |

ANGULAR POSITION

0.2 rad |

FPL EMG

100 ms

FIGURE 28. Stimulation of the median nerve to evoke a
maximal e.m.g. response in the FPL muscle produced the
greatest velocity and acceleration for an initial angle of
approximately 0.6 rad (dotted lines). Peak velocity and
acceleration declined progressively as the initial angle
decreased. The reduction in peak acceleration was not as
great as when the initial angle increased (Figure 27).

pressure of the stimulating electrode against the nerve. The peak flexion velocity, on the other hand, decreased systematically as the angle increased (Figure 27) or decreased (Figure 28) from the middle position (0.6 rad). It changed by about 30% with respect to its largest value. Peak angular acceleration was affected in the same way as velocity, although the decrements were greater as the initial angle increased (Figure 27) from the mid-position than as it decreased (Figure 28).

Figure 29 shows that velocity and acceleration increase in proportion to the magnitude of the FPL e.m.g. for a given initial angle. At lower levels of stimulation the e.m.g. begins later and has a slower rate of rise. This is also reflected in the delay to movement onset. As the e.m.g. increases movement begins sooner and accelerates more rapidly.
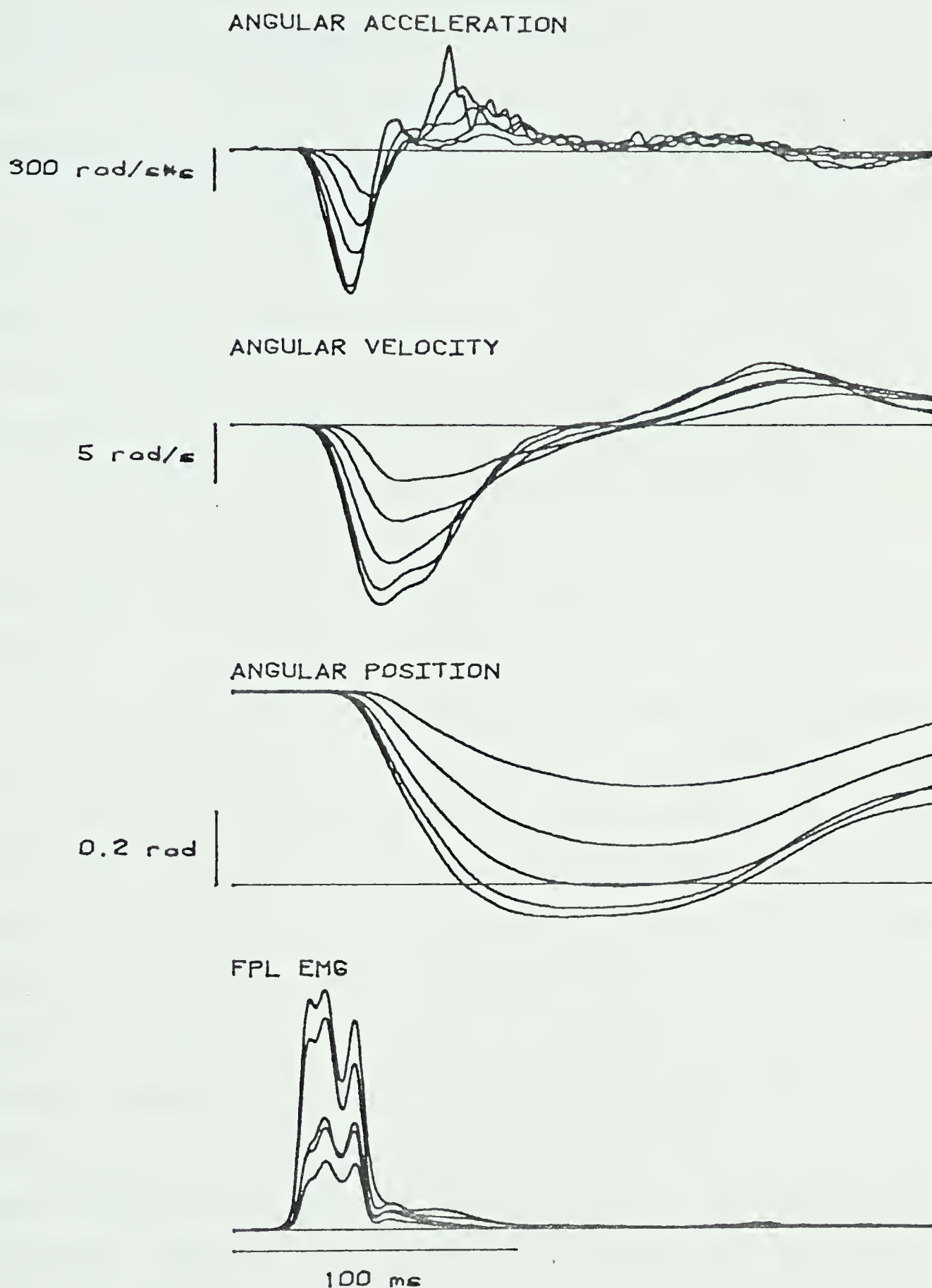
FIGURE 29. Increasing the strength of stimulation of the median nerve while keeping the initial angle fixed produced progressive increases in peak velocity and acceleration. The delay between onset of movement and FPL e.m.g. onset declined to about 20 ms as stimulus strength increased.

VI. DISCUSSION

In this chapter the results of experiments in the preceding chapters and their implications in the motor control of 'ballistic' movements will be discussed.

'Ballistic' movements are employed in a number of tasks where the objective is to impart an impulse force to an object. The golf swing, tennis serve, baseball pitch, and volleyball spike are examples. Sometimes 'ballistic' movements are arrested by an obstacle. This occurs in striking a nail with a hammer, playing a keyboard or percussion instrument, fingering a woodwind or brass instrument, typing or producing certain consonant sounds in speech.

The conclusions of this study have implications for the regulation of velocity in these types of movements. Although only single-joint movements of the thumb were investigated, where the moment of inertia is relatively low, some of the principles underlying the organization of 'ballistic' thumb movements may carry over to movements where inertia is greater or where there is motion at several joints. For example, Marsden et al. (1983) have shown that there are similarities in the regulation of velocity in 'self-terminated' thumb and elbow movements while Wadman et al. (1979, 1980) have observed similar velocity-amplitude relationships in rapid single-joint and two-joint movements.

This study has focused on the way in which velocity is regulated and how it is judged by subjects executing

'ballistic' movements, in particular, what aspects of sensation are used as criteria in reproducing peak velocity.

The control strategies which subjects employed in regulating amplitude or velocity in reciprocating movements resulted in a linear relationship between peak velocity and movement amplitude. Although this relationship would follow from pulse-height regulation of the neural input, the data indicate that in general, subjects also modulated the duration of the neural impulse. Thus, in many cases movements of different amplitudes were scaled in time.

The extent to which the relationship between peak velocity and movement amplitude could be altered by changing task conditions or movement frequency indicates that there are limitations within which the central nervous system is constrained to operate when executing 'ballistic' movements.

The peak velocity of a 'ballistic' movement is apparently not judged on the basis of peripheral sensory information, but according to an internal model of the movement which is based more directly on the motor command.

A. Muscle Function in Reciprocating Movements

Consider first the roles that the antagonistic muscles play in the generation of 'ballistic' reciprocating movements. These movements were generated by two consecutive force impulses, the first delivered to the EPL muscle, the second to the FPL muscle. The function of the impulse delivered to the EPL muscle was obviously to initiate

extension, causing the distal phalanx of the thumb to move until resistive forces brought its velocity back to zero. The impulse delivered to the FPL muscle may have had more than one function though. In addition to producing flexion of the interphalangeal joint of the thumb, the flexor impulse may have acted to brake extension. However, there are several arguments against such a role for the flexor impulse.

Careful examination of Figure 5 shows that activity in FPL begins only about 25 ms before the angular velocity reaches zero. Figure 29 shows that the delay between the onset of FPL e.m.g. and the onset of movement is approximately 20 ms when the median nerve branch to FPL is stimulated supramaximally. The delay increases to 30 ms as the strength of the stimulus is reduced. Thus, in Figure 5 extension velocity may have already decreased to zero by the time the FPL muscle began to actively generate force.

A simulation was carried out using the model of Oğuztöreli and Stein (1982) to determine what role the flexor might play in braking. The implementation of this model is outlined in Appendix 1. In one simulation the extensor muscle alone was activated for 120 ms (Figure 30, trajectory A). In a second simulation extensor activation was followed immediately by flexor activation (Figure 30, trajectory B). Activation of the flexor had almost no effect on the extension phase of the simulated movement. According to the simulation, passive visco-elastic forces developed by
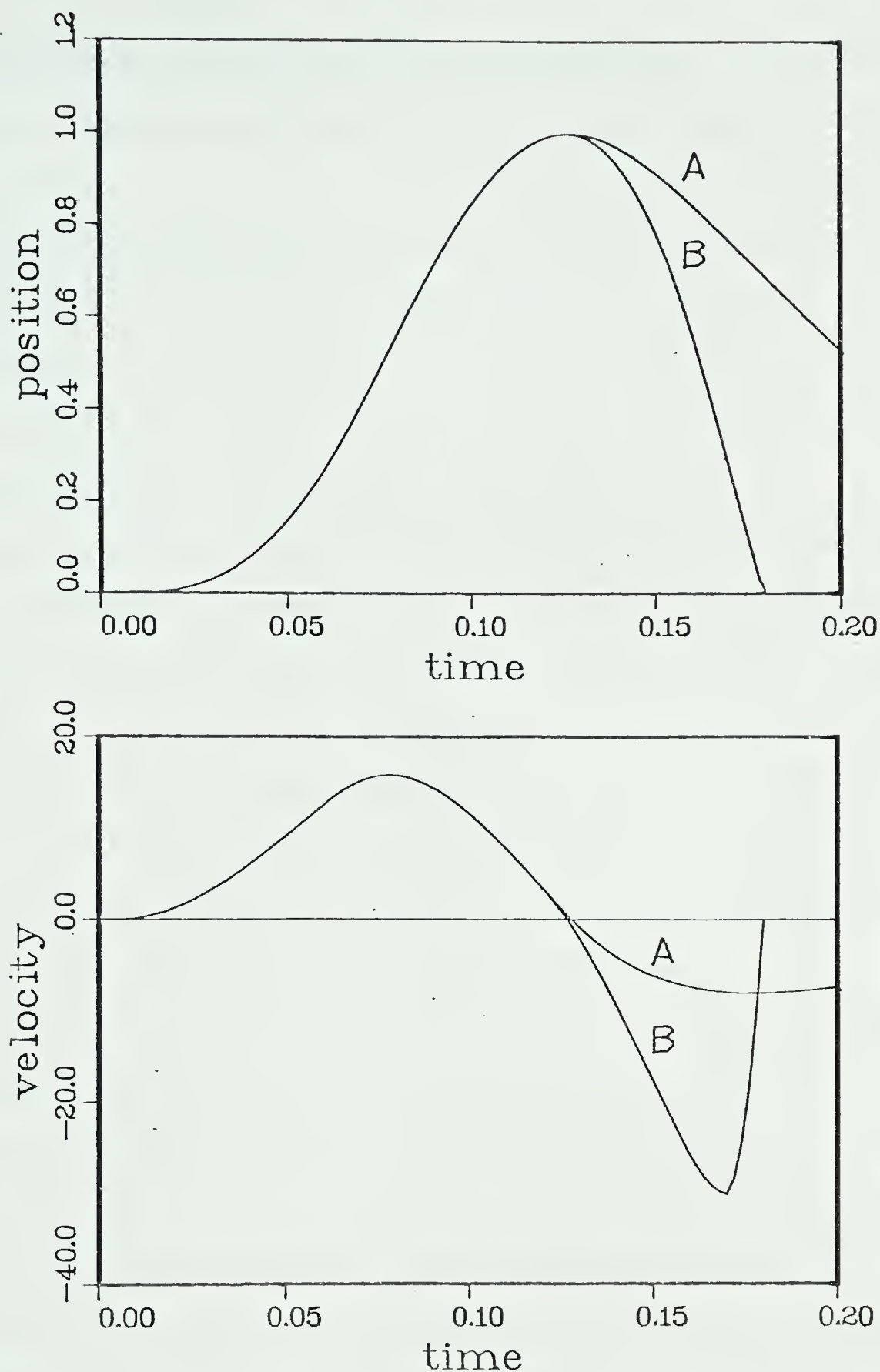
FIGURE 30. Position and velocity trajectories of simulated reciprocating movements using the model described in Appendix 1. Trajectories labeled A were obtained when only the extensor muscle was activated. Those labeled B were obtained when extensor activation was followed immediately by activation of the flexor muscle. Velocity trajectory B returns abruptly to zero during flexion when contact is made with the mechanical stop (zero position). There is almost no difference in the extension phase of trajectories A and B.

the muscles are sufficient to stop extension. Therefore, the FPL force impulse need play little, if any role in braking extension.

The simulation also shows that only one process (extensor contraction) is responsible for producing extension velocity, while two processes (return of the stretched flexor to its relaxed length and flexor contraction) sum to produce flexion velocity.

Because the mathematical model is linear, it predicts a linear relationship between peak extension velocity and movement amplitude when the amplitude of the neural input is scaled in a linear fashion (pulse-height regulation). Thus, if subjects had regulated only the amplitude of the neural input, but not its duration, their movements would have been constrained a priori to exhibit a linear relationship between peak velocity and movement amplitude.

The fact that e.m.g. burst duration could be reduced by increasing movement frequency (Figure 8) and that burst duration tended to increase with movment amplitude (Figures 13, 14 and 20) indicates that burst duration is regulated by the central nervous system. While the primary determinant of the observed linear relationship between peak velocity and movement amplitude is probably pulse-height regulation of the neural input, there is also pulse-width modulation which is dependent on movement amplitude.

B. Muscle Function in Flexion Movements

The flexion movement was in some respects similar to the flexion phase of a reciprocating movement. However, there were also some major differences. Since flexion was not immediately preceded by phasic excitation of the antagonist muscle, any constraints which would have been imposed by the need to coordinate reciprocal muscle activity were eliminated.

Furthermore, there was little room for flexibility in strategy in the flexion movements since both amplitude and peak velocity were specified. Peak velocity could be regulated only by altering muscle activation. The linear relationships observed between peak flexion velocity and FPL e.m.g. (Figure 19) might be expected. They are consistent with the Oğuztöreli and Stein muscle model which predicts such linear relationships between peak velocity and neural input when movement amplitude is constant, provided it is large enough to allow peak velocity to occur before impact is made with the mechanical stop.

The results of the experiments involving single flexion movements show that 'ballistic' movements can be performed over approximately a three-fold range of velocities without altering movement amplitude. Since rate of rise, amplitude and integrated area of the FPL e.m.g. burst all correlate linearly with peak angular flexion velocity while burst duration correlates relatively poorly, velocity must be regulated by a pulse-height command to the motoneuron pool.

Similar correlations between burst amplitude or integrated area and peak velocity have been reported by a number of investigators (Bouisset and Lestienne, 1974; Lestienne, 1979; Hallett and Marsden, 1979; Brown and Cooke, 1981; Hoffman and Strick, 1982; Marsden et al., 1983). Zangemeister, et al. (1982), investigating the control of head rotation, found that the rate of rise of the e.m.g. burst correlated as well with peak acceleration as did the burst amplitude. It follows that a strong correlation would also have existed between rate of rise of the e.m.g. burst and peak velocity, as in the present study.

C. Movement Control Theories

The simulations do not provide information about the control strategies of the central nervous system but only indicate what the response of the antagonistic muscle system will be, given a particular neural input.

Given that the amplitude and integrated area of the e.m.g. burst were better correlated with the parameters of movement than the durations of the burst (Figures 13, 14 and 19), it follows that during 'ballistic' movements the initial (rising) phase of the e.m.g. was more important than the duration of the burst in determining the movement trajectory. Several of the control strategies which were described in the Introduction focus on these initial events and are discussed below. Cooke (1980), Fel'dman (1980b) and Bizzi et al. (1982) all suggest that rapid single-joint

movements are initiated by an abrupt (step-like) shift in the length-tension characteristics of antagonistic muscles. Their ideas differ though, as to the particular aspect of the length-tension characteristic which is being regulated. Cooke (1980) proposed that there was a shift in muscle stiffness, Fel'dman (1980b) claimed that the invariant characteristic or the muscle zero length shifted, while Bizzi et al. concluded that it was the equilibrium point that shifted.

Strictly speaking there is no equilibrium point in a 'ballistic' movement since it consists only of an impulse phase. There is only a transient response which eventually returns to the initial position if muscle activity returns to its pre-movement level. This is evident from the simulation discussed above (Figure 30). Furthermore, the changes in muscle activity and force are pulsatile rather than step-like. However, it is still reasonable to ask whether the characteristics of the force could result from a shift in muscle stiffness or zero length.

Cooke's model characterized an antagonistic pair of muscles as a single damped spring. This is quite restrictive as was pointed out in the Introduction. The model can be made more realistic by using two springs acting in opposition to one another. The differential equation which describes the model can be solved analytically. It is possible, therefore, to test whether step changes in muscle stiffness can result in a linear relationship between peak

velocity and movement amplitude. The general solution to the differential equation is given in Appendix 2. It shows that even when simplifying assumptions are made, peak velocity will be proportional to the square root of the movement amplitude. Under a log-log transformation the slope of peak velocity versus movement amplitude will be 0.5 if the relationship is square root and 1.0 if it is linear. Log-log transformation of the data in Part B of Chapter V (for all six subjects, including those in Figures 10 and 11) gives slopes for the relationships between peak extension velocity and movement amplitude that are closer to 1.0 than 0.5 (0.80±0.15[s.d.], n=18) so it is unlikely that 'ballistic' movements are regulated by step-like changes in muscle stiffness only. Measurements of torque versus angular position (Cooke, 1982) indicated that net joint stiffness remained relatively constant for movements of progressively increasing amplitude, providing empirical evidence that changes in muscle stiffness are unlikely to be the mechanism regulating the velocity of rapid movements.

According to Fel'dman, rapid movements are initiated by sudden shifts in the zero length of the agonist muscle (Fel'dman, 1974b). He gives only a qualitative description of this process. To test whether his theory predicts a linear relationship between peak velocity and movement amplitude it was applied with certain simplifying assumptions.

First, consider Fel'dman's invariant characteristics. They all have downward convexity, implying that muscle stiffness is reduced as muscle torque decreases (for a given muscle zero length). As Fel'dman (1966a) argued, this is probably a property of the integrated reflex inputs to alpha motoneurons. In this discussion, muscle stiffness will be assumed to be constant for all muscle zero lengths. Fel'dman (1966a) drew attention to the fact that the shape of the invariant characteristics was relatively independent of muscle zero length. Making the further assumption that the stiffness is constant can be justified if there is little difference between initial and final muscle torques, since the slopes of the the initial and final invariant characteristics, i.e. the initial and final stiffnesses will then be approximately equal.

Fel'dman does not give an explicit mathematical description of muscles in his theory, although he characterizes them as damped springs. The differential equation used to quantify Cooke's model in Appendix 2 describes damped springs. It has been appropriately modified in Appendix 3 to provide a mathematical description of Fel'dman's theory, incorporating the simplifications discussed above. The solution shows that Fel'dman's theory predicts a linear relationship between movement amplitude and the shift in muscle zero length. Since peak velocity is directly proportional to movement amplitude in such a system, provided stiffness and damping remain constant, it

follows that Fel'dman's theory predicts a linear relationship between peak velocity and the shift in muscle zero length.

Fel'dman's theory is also compatible with the observed linear relationship between peak velocity and muscle activity (Figures 13, 14 and 19) since muscle zero length is directly related to the excitability of the motoneuron pool.

The equilibrium shift hypothesis of Bizzi et al. (1982) is equivalent to either one or the other of the above hypotheses (or some combination of the two) if antagonistic muscles are characterized as damped springs. In a system of springs there are only two parameters which can be varied to change the equilibrium point, namely the spring stiffness and the zero length. Therefore, an abrupt shift in the equilibrium point could produce a linear relationship between peak velocity and movement amplitude if it were brought about by a shift in zero length.

The symmetric impulse-variability theory of Meyer et al. (1982) cannot be applied in its entirety to the movements of this study because these movements did not require a decelerative force impulse for braking. The flexion movements consisted of a single accelerative force impulse while the reciprocating movements were effectively composed of two oppositely directed accelerative force impulses which were delayed sufficiently with respect to each other that they did not overlap. Thus, only those aspects of the theory which do not require the incorporation

of a decelerative force impulse can be applied. The fundamental principles of time and force scaling do fall into this category.

As Figure 12 indicates, reciprocating movements were often time scaled, in contrast to the constancy of movement time which Freund and Büdingen (1978) had reported. However, this was not strict time scaling in terms of its definition in the symmetric impulse-variability theory. According to the theory, when movements are scaled in time only, acceleration will initially be higher for shorter (smaller amplitude) movements than longer (larger amplitude) movements (Figure 2). This will produce a steeper initial slope for the angular position of small amplitude movements. It is clear from Figure 12 that the initial slope is generally smaller for small amplitude movements and may be relatively invariant for movements of intermediate and large amplitude.

The observed trajectories are more likely produced by a combination of time and force scaling since force scaling results in initial accelerations which are higher for movements of larger amplitude (Figure 2).

In flexion movements there was often a remarkable similarity in the initial phase of the velocity trajectories which appeared to be relatively independent of initial angle or target velocity (Figures 16, 17 and 18). Although it may not be obvious how this could arise from a simple combination of time and force scaling, it is a feature of

the Oğuztöreli and Stein model. Whenever the initial slopes of the neural inputs are the same, velocity trajectories will coincide. This is a consequence of the linearity of the model.

Therefore, the initial similarity of velocity trajectories during 'ballistic' movements is most adequately explained if patterns of motor unit recruitment are taken into account. The similarity of the rising phase of FPL e.m.g. bursts when velocity trajectories coincide (Figure 18) suggests that the shape of the accelerative force impulse is governed by the manner in which motor units are recruited. During 'ballistic' contractions motor units follow an orderly pattern of recruitment and are activated more often as the strength of the contraction increases (Desmedt and Godaux, 1978b). They rarely fire more than three times during the course of a 'ballistic' contraction though. Therefore, there may be a saturation in the firing of low-threshold motor units which when combined with their orderly recruitment and the force-velocity properties of muscles, limits the initial acceleration and causes the initial coincidence of all velocity trajectories beyond the saturation point.

The similarity in the initial phase of the velocity trajectories opposes the notions that 'ballistic' movements of greater velocity or amplitude are produced by greater shifts in either the muscle zero length or stiffness. Both predict that initial acceleration should be linearly scaled

with the magnitude of the shift and hence should increase for higher peak velocities (Appendices 2 and 3). Instead, it appears that the initial shift in the length-tension characteristics of the muscle is often very similar for all 'ballistic' movements and that there is a later shift which depends on amplitude or peak velocity.

D. Independent Regulation in Reciprocating Movements

As was mentioned above, both the extension and flexion phases of 'ballistic' reciprocating movements were similarly regulated. It is noteworthy, therefore, that a certain degree of independent control of the two phases could be achieved by varying the amplitude target while keeping the velocity target (peak flexion velocity) fixed. In Figure 10 peak extension velocity retained a high linear correlation with amplitude and the relationship showed little change in slope while peak flexion velocity became relatively poorly correlated with amplitude with a dramatic reduction in the slope of the relationship. Hoffman and Strick (1982) have seen similar independence of agonist and antagonist muscle activity in 'self-terminated' movements.

Nonetheless, the central nervous system apparently prefers to use a strategy which links the amplitude and velocity of 'ballistic' movements whenever possible. Thus, when the amplitude target was fixed while the velocity target varied, both extension and flexion velocities were again similarly linked to amplitude (Figure 11). The reduced

linear correlation was probably a combination of the demands of the task, i.e. forcing the velocity higher for a given movement amplitude, and the attempt to disrupt the mode of velocity regulation preferred by the central nervous system.

Since the slope of the relationship between peak flexion velocity and movement amplitude could not be increased by keeping the amplitude target fixed it is likely that in linking peak velocity and movement amplitude, subjects were employing a strategy which either maximized peak velocity or optimized the process of regulation.

The fact that there was a tendency for the slope of peak extension velocity versus amplitude to be reduced while the slope of peak flexion velocity versus amplitude increased in going from the amplitude to the velocity task (Figures 10 and 11) suggests that the focus of the central strategy shifted according to the parameter being displayed during the task, allowing the peak velocity to increase for any given movement amplitude.

E. Control of Rhythmic Reciprocating Movements

Individual reciprocating movements which were embedded in a rhythmic sequence had all of the characteristic features of the movements described above, but because the sequence could be performed at different tempos it was possible to observe temporal scaling features which were relatively independent of movement amplitude.

Stetson (1905) and Stetson and McDill (1923) studied rhythmic 'ballistic' movements without the aid of e.m.g.'s. They had subjects perform rhythmic up-and-down movements of the upper limb. They called the up-stroke of these movements, the back-stroke, and the down-stroke, the beat-stroke, in analogy to the movements used in percussion or music conduction. In the reciprocating movements of the present study extension corresponded to the back-stroke and flexion to the beat-stroke.

Stetson (1905) suggested that braking of the back-stroke might be achieved passively without requiring activation of the muscles generating the beat-stroke. This was borne out in the present study, as discussed in the first section of this chapter.

He showed that the average velocity of the beat-stroke was dependent on the length of the stroke and not on the tempo of the rhythm and that its duration was less dependent on the tempo than the duration of the back-stroke. The present study verifies these findings. There was a linear relationship between peak flexion velocity and amplitude during the beat-stroke which was relatively invariant over a range of tempos and as Figures 8 and 9 demonstrate, the duration of the EPL e.m.g. burst (back-stroke) was more strongly dependent on tempo than the duration of the FPL e.m.g. burst (beat-stroke).

Stetson and McDill (1923) presented evidence that the inter-movement interval was longer than the movement

duration and that when tempo increased this interval was reduced by a greater proportion than the movement duration. This is again evident from Figures 8 and 9.

The studies of Stetson (1905) and Stetson and McDill (1923) did not report any breakdown of the relationship between velocity and amplitude at fast tempos. However, they did not examine a very wide range of tempos and movement amplitudes. This breakdown, which is illustrated in Figure 7, was most likely a consequence of insufficient time between movements to allow the central nervous system to prepare an organized movement structure.

One striking feature about the timing of muscle activity is the relative duration of EPL and FPL bursts. The EPL burst seems to occupy the entire inter-movement interval and extends well into the extension phase of movement (Figure 6). It was often twice as long as the FPL burst. Yet, there was apparently a net force in the flexor direction for about half of the inter-movement interval since angular position declined during that period (Figure 8). Onset of extension was delayed at least 80 ms from the onset of EPL activity in some cases. This suggests that following flexion the EPL muscle is relatively inefficient in generating force. The inability of the EPL muscle to generate force quickly following flexion may, therefore, be a significant factor in limiting the frequency of reciprocating movments.

As noted earlier, the linear relationship between peak velocity and movement amplitude remained relatively invariant over a range of tempos. Since e.m.g. burst duration decreased as movement frequency increased (Figure 9), there had to be a compensatory increase in e.m.g. burst amplitude (Figure 8). Unlike the rhythmic forearm movements which Fel'dman (1980a) observed, movement amplitude was not reduced as frequency increased because the task required that the same peak flexion velocity be maintained. Peak velocity was linked to movement amplitude, hence amplitude did not decline. At very fast tempos it likely became increasingly more difficult to provide the necessary compensation in e.m.g. burst amplitude as burst duration shortened, causing performance to deteriorate.

F. Judgment and Perception of Velocity

The velocity mismatch, induced by instructing subjects to change the angular position of movement initiation (Figures 16 and 20), appears to result from the change in position, rather than any accompanying change in movement amplitude, since it does not occur when amplitude increases while initial angle is fixed (Figure 20). It might be argued that no mismatch occurs in the latter case because the central nervous system utilizes the same voluntary motor command during the larger and smaller amplitude movements, the only difference being that the smaller amplitude movement is terminated by the mechanical stop at an earlier

point in the movement trajectory. As Figure 20 demonstrates this is not the case. The FPL e.m.g. burst duration is longer for the larger amplitude movement although the rising phases of the e.m.g.'s are very similar.

Velocity undershoots or overshoots seen when changing both initial angle and amplitude were generally accompanied by decreases (Figure 16) or increases (Figure 17) in the respective FPL e.m.g. burst amplitudes. Yet, when amplitude was held constant and initial angle increased, overshooting occurred even though FPL e.m.g. bursts often differed little (Figure 21). Both muscle length-tension characteristics and muscle activation strength were, therefore, factors responsible for the overshoot.

The velocity and acceleration trajectories resulting from supramaximal stimulation of the FPL nerve (Figure 27) have very similar initial profiles, not unlike the coincidence of velocity trajectories illustrated in Figures 16 and 17. There is a major difference, of course, in that synchronization of the entire motoneuron pool by peripheral nerve stimulation produces higher accelerations. The greatest acceleration is achieved for an initial angle of approximately 0.6 rad. Both peak acceleration and velocity diminish when the angle is incremented or decremented by approximately 0.1 rad (Figure 27).

Thus, when a voluntary movement is initiated from an angle of 0.75 rad it first passes through a region where acceleration is an increasing function of muscle length,

whereas when initiated from an angle of 0.55 rad it is entirely within a region where acceleration is a decreasing function of muscle length. This would explain how a movement initiated from an angle of 0.75 rad and having the same amplitude and pattern of muscle activation as a movement initiated from an angle of 0.55 rad (as judged from the FPL e.m.g. burst), could result in a higher peak velocity.

Subjects apparently were unaware of the added acceleration generated by passing through this region of increasing acceleration and did not adjust the voluntary motor command to the FPL muscle. FPL e.m.g. records indicate that muscle activation either remained the same or was increased. An increase was not necessarily due to an increment in voluntary drive to the motoneuron pool. Instead, it may have reflected an increased excitability of the motoneuron pool due to reflex inputs from muscle stretch receptors which increased their firing rates in response to the additional passive stretching of the FPL muscle (Hulliger et al., 1982).

Therefore, in matching peak velocities, subjects were probably matching the voluntary motor command. This is even more evident in the mismatching which occurred when loading conditions changed. The differences between the peak velocities of loaded and unloaded movements were often very large because subjects did not compensate with appropriate changes in muscle activation (Figures 22 and 23).

Although subjects were undoubtedly receiving sensory input which could have provided a fairly reliable measure of peak velocity, they apparently did not use it matching velocity when visual feedback was withheld. For example, the force of impact with the mechancial stop or the time taken to reach the stop following movement initiation should have been the same for movements of the same peak velocity initiated from different angles but having the same amplitude. Had subjects been judging velocity according to impact force or movement time, it is unlikely that they would have overshot the velocity they were attempting to match.

There was one subject who claimed to be matching movement times. Her results bore this out. She was less prone to overshooting when amplitude was fixed and initial position changed than other subjects, but was more prone to overshooting when initial position was fixed and amplitude increased. She was also one of the two subjects who compensated adequately when loading was varied.

One of the suprising findings of this study was that subjects did not appear to base judgments of peak velocity on signals from peripheral sensory receptors. Cafarelli and Bigland-Ritchie (1979) arrived at a similar conclusion from the results of force matching experiments. When subjects were asked to match forces in contralateral muscle pairs where muscle lengths differed, they found that e.m.g.'s were much better matched than muscle forces, suggesting that

central mechanisms were more important than peripheral
mechanisms for force sensation.

From the present study it appears that the internal
reference frame used by the central nervous system is highly
dependent on the state of the peripheral nervous system. The
representation or model of a 'ballistic' movement in the
internal reference frame is a combination of sensations
derived from peripheral sensory receptors and voluntary
motor commands. Fel'dman and Latash (1982) describe how this
process can lead to kinesthetic illusions in relation to
joint position sense. Similar phenomena may lead to the
misjudgments of velocity during 'ballistic' movements
described in the present study.

The mapping of the external reference frame to the
internal reference frame is probably peculiar to each
individual, particularly in terms of the peripheral sensory
cues which are selected for attention. As a task is
practiced, an internal model of the movement is memorized
and it is this model that is recalled and reproduced. It is
not velocity, as measured in the external reference frame
which is being matched, but an internal representation of
the movement which may or may not incorporate a peripheral
sensory representation of velocity.

When the external reference frame is manipulated
without providing an opportunity for recalibration of the
external to internal mapping, i.e. withholding knowledge of
results, a subject will match the internal representation of

those aspects of the movement which reproduce the sensation that constitutes the internal model of the movement (Goodwin et al., 1976). For 'ballistic' movements this sensation is related more directly to the voluntary motor command than to peripheral sensory feedback.

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Appendix 1

The antagonistic muscle model of Oğuztöreli and Stein (1982) was implemented using parameters which made the flexor muscle 2.5 times as strong as the extensor. This ratio was chosen on the basis of the maximal forces which could be generated by extension and flexion of the interphalangeal joint of the thumb. The model incorporated a small inertial load (0.05 kg) which corresponded to the combined mass of the distal phalanx of the thumb and the cage in which it was clamped. There was also a relatively large viscous load (100 N·s/m), chosen because it produced simulated trajectories which were similar to observed movement trajectories. The extensor and flexor muscles were activated reciprocally using a neural input which was triangular in shape. The triangle was symmetric, peaking at 60 ms for the extensor muscle and 40 ms for the flexor. These values were chosen on the basis of EPL and FPL e.m.g. burst profiles recorded during 'ballistic' reciprocating movements. The values of the muscle parameters used in the simulations were based on the 'standard' values used by Oğuztöreli and Stein (1982). They are listed below.

Extensor active state viscosity	40 N·s/m
Flexor active state viscosity	40 N·s/m
Extensor series elastic stiffness	2200 N/m
Flexor series elastic stiffness	5500 N/m
Extensor parallel elastic stiffness	880 N/m
Flexor parallel elastic stiffness	2200 N/m
Rate constant for active state decay	$30s^{-1}$

In order to apply the results of this simulation directly to movements of the interphalageal joint of the

thumb it is necessary to show that rotation of this joint is directly proportional to linear movement of the EPL and FPL muscles. The distal phalanx of the thumb is modeled as as a block sliding along a curved surface in response to forces exerted by the EPL muscle in one direction and the FPL muscle in the opposite direction (Figure 31). Muscle forces are transmitted to the block by means of long tendons which follow the contours of the joint. Change in joint angle is well-approximated as a linear function of change in length of either muscle, as shown below.

For the extensor muscle (Figure 28A) change in muscle length is directly proportional to change in the length of tendon lying along the outer contour of the joint. This change in length will be directly proportional to the change in joint angle.

It is not as obvious that changes in joint angle can produce proportional changes in the length of tendon lying along the inner contour of the joint, i.e. the flexor tendon (Figure 31B). Equation 1.1 gives the relationship between the angle θ and the length of tendon ℓ.

$$\ell = r\sqrt{[a^2 + 2(2+a)(1 - \cos\theta) + 2a\sin\theta]} \qquad 1.1$$

where r is the radius of curvature of the interphalangeal joint and a is a scaling factor that depends on the point of insertion of the tendon. This relationship is plotted for a range of values of a in Figure 32. The linear correlation coefficient was greater than 0.999 in every case. Therefore, the error made in assuming that the relationship is linear
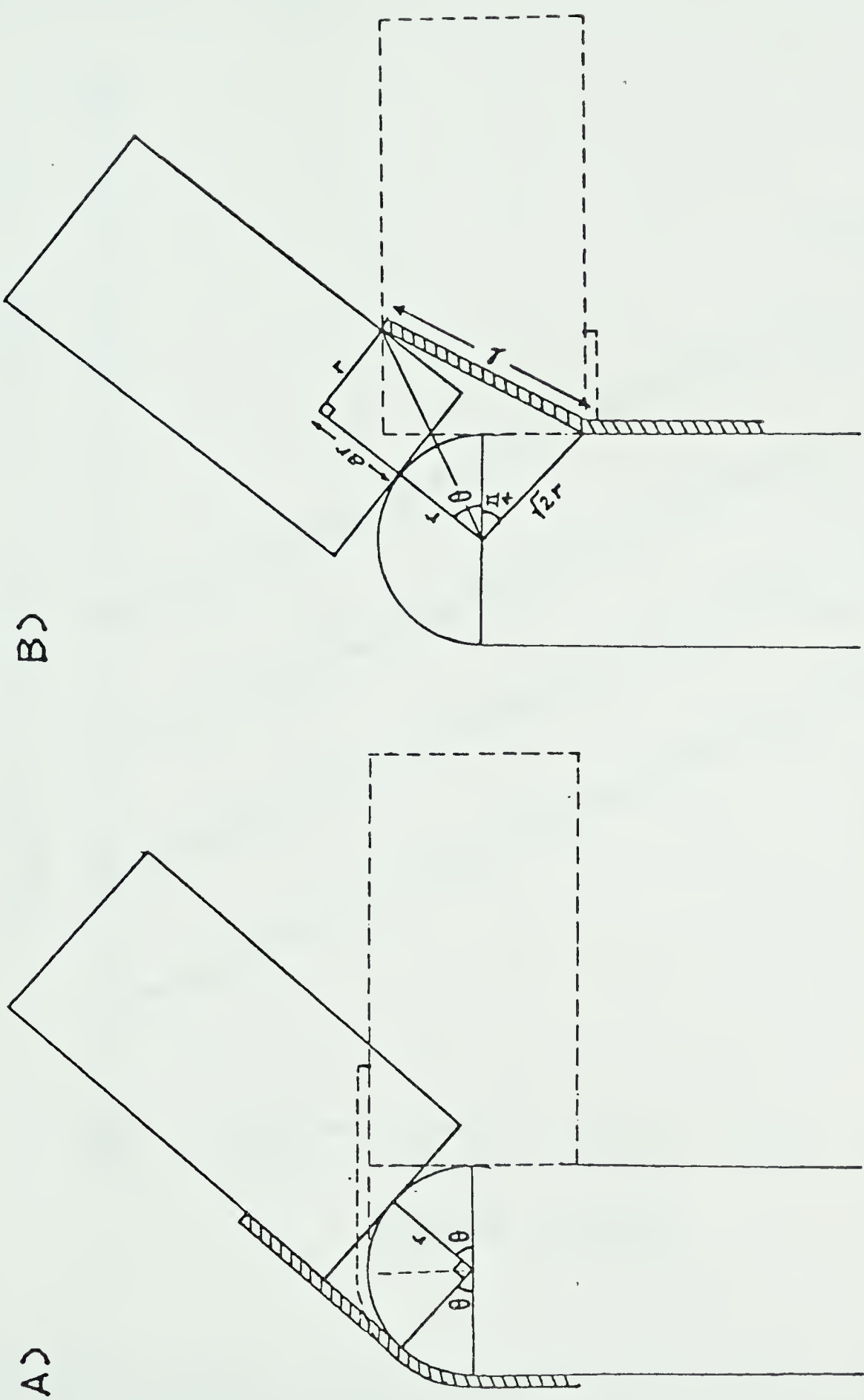
FIGURE 31. Simple biomechanical model of the interphalangeal joint of the thumb. A) Hatched portion represents the extensor tendon. The tendon length following the contour of the joint is a function of the angle of rotation θ and the radius of curvature r. B) Hatched portion represents the flexor tendon. The tendon length following the contour of the joint is a function of θ, r and the point of attachment of the tendon represented by the quantity ar (Equation 1.1).
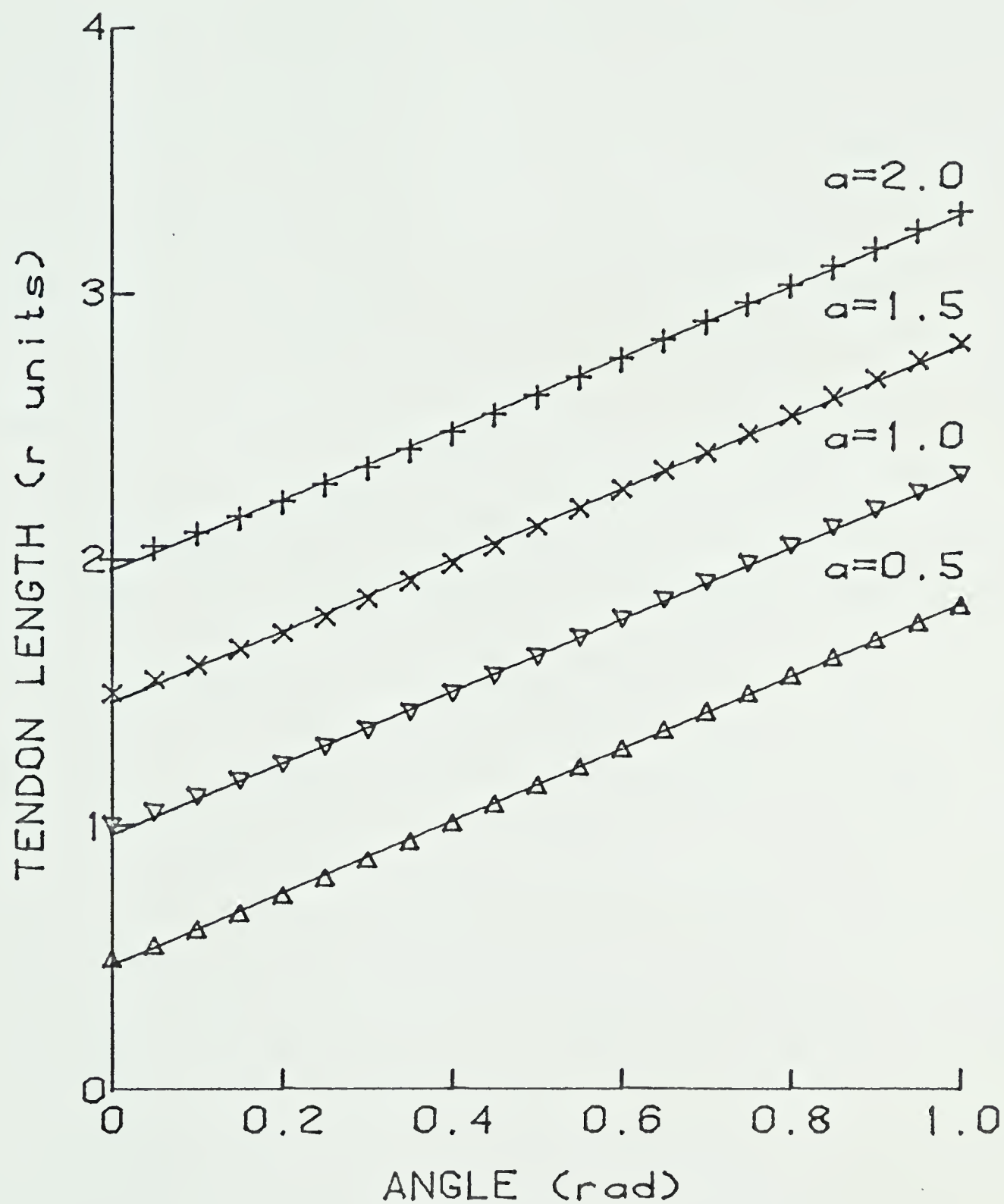
FIGURE 32. Plots of Equation 1.1 over the angular region 0 to 1 rad for several values of the scaling factor *a*. The linear correlation correlation coefficients are all greater than 0.999.

over the angular range 0 to 1 rad, i.e. the region to which
the movements of this study were confined, is neglibible.

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Appendix 2

Figure 33 shows a simple damped spring model for a pair of antagonistic muscles. This model will be used to determine whether a step change in stiffness can produce a linear relationship between peak velocity and movement amplitude. The equation of motion is given below.

$$m\ddot{x}(t) + \eta\dot{x}(t) + (k_1 + k_2 + \Delta_1(t) + \Delta_2(t))x(t) - \Delta_1(t)x_1 - \Delta_2(t)x_2 = 0 \qquad 2.1$$

$$\Delta_i(t) = 0 \qquad\qquad t \le 0, \quad i = 1, 2 \qquad\qquad 2.2$$
$$\quad = k_i' - k_i \qquad t > 0$$

$$k_1 x_1 + k_2 x_2 = 0 \qquad\qquad 2.3$$

where k_i are the initial stiffnesses, k_i' the final stiffnesses and the positions x_1 and x_2 correspond to the respective zero lengths. Using 2.2 and the fact that $x = 0$ at $t = 0$, 2.1 becomes

$$m\ddot{x}(t) + \eta\dot{x}(t) + (k_1' + k_2')x(t) - \Delta_1(t)x_1 - \Delta_2(t)x_2 = 0 \qquad 2.4$$

Let $\beta = \eta/2m$ and $\omega_0 = \sqrt{(k_1' + k_2')/m}$ then taking the Laplace transform of 2.4 we get

$$mX(s)[s^2 + 2\beta s + \omega_0^2] - 1/s[(k_1' - k_1)x_1 + (k_2' - k_2)x_2] = 0 \qquad 2.5$$

Using 2.3 and simplifying we get

$$X(s)[s^2 + 2\beta s + \omega_0^2] = 1/s[(k_1'x_1 + k_2'x_2)/m] \qquad 2.6$$

Let $\alpha = (k_1'x_1 + k_2'x_2)/m$ then

$$X(s) = \alpha/[s(s^2 + 2\beta s + \omega_0^2)] \qquad 2.7$$

Inverting $X(s)$ we get

$$x(t) = \alpha/\omega_0^2\{1 - exp(-\beta t)[cosh\omega t + (\beta/\omega)sinh\omega t]\} \qquad 2.8$$

where $\omega = \sqrt{(\beta^2 - \omega_0^2)}$. Differentiating 2.8 we get

$$\dot{x}(t) = \alpha/\omega[exp(-\beta t)sinh\omega t] \qquad 2.9$$
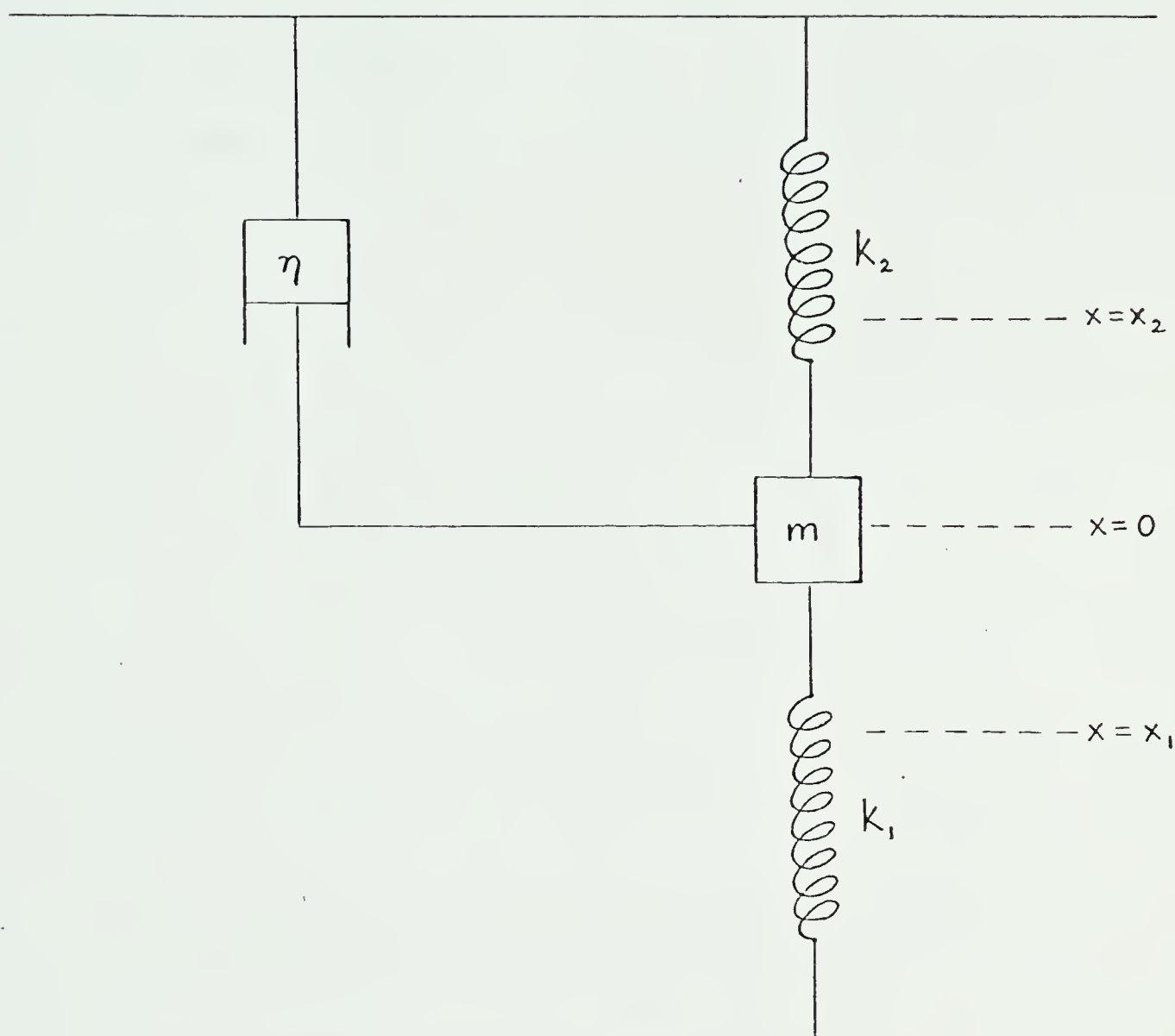
The maximum value of 2.8 is given by

FIGURE 33. Damped spring model of antagonistic muscle pair. The spring stiffnesses are k_1 and k_2, the respective positions of zero length are x_1 and x_2, m is the mass and η the coefficient of viscosity. The equilibrium point is x=0, i.e. $k_1 x_1 + k_2 + x_2 = 0$.

$$x_m = \alpha/\omega_0^2 \qquad\qquad 2.10$$

The maximum value of 2.9 is given by

$$\dot{x}_m = \omega_0 exp\{-(\beta/2\omega)ln[(\beta+\omega)/(\beta-\omega)]\}x_m \qquad\qquad 2.11$$

It is obvious that there is no simple linear relationship between peak velocity $\dot{x}_m$ and amplitude x_m. Even if we make the simplifying assumptions that k_2' is zero and that $\beta=\omega_0$ (critical damping), $\dot{x}_m$ is proportional to the square root of x_m.

Appendix 3

The model of Figure 33 will be used to establish whether a step change in zero length can produce a linear relationship between peak velocity and movement amplitude. The equation of motion is given below.

$$m\ddot{x}(t)+\eta\dot{x}(t)+(k_1+k_2)x(t)-k_1(x_1+\Delta_1(t))-k_2(x_2+\Delta_2(t))=0 \qquad 3.1$$

$$\begin{aligned}\Delta_i(t)&=0 \qquad &t\le 0, \quad i=1,2 \\ &=x_i{}'-x_i \qquad &t>0\end{aligned} \qquad 3.2$$

$$k_1x_1+k_2x_2=0 \qquad 3.3$$

where x_i are the positions corresponding to the initial zero lengths, $x_i{}'$ to the final zero lengths and k_1 and k_2 are the respcetive stiffnesses. Using 3.3 to simplify 3.1 we get

$$m\ddot{x}(t)+\eta\dot{x}(t)+(k_1+k_2)x(t)-k_1\Delta_1(t)-k_2\Delta_2(t)=0 \qquad 3.4$$

Let $\beta=\eta/2m$ and $\omega_0=\sqrt{(k_1+k_2)/m}$ then taking the Laplace transform of 3.4 we get

$$mX(s)[s^2+2\beta s+\omega_0{}^2]-1/s[k_1x_1{}'+k_2x_2{}']=0 \qquad 3.5$$

$$X(s)[s^2+2\beta s+\omega_0{}^2]=1/s[(k_1x_1{}'+k_2x_2{}')/m] \qquad 3.6$$

Let $\alpha=(k_1x_1{}'+k_2x_2{}')/m$ then

$$X(s)=\alpha/[s(s^2+2\beta s+\omega_0{}^2)] \qquad 3.7$$

Inverting $X(s)$ we get

$$x(t)=\alpha/\omega_0{}^2\{1-exp(-\beta t)[cosh\omega t+(\beta/\omega)sinh\omega t]\} \qquad 3.8$$

where $\omega=\sqrt{(\beta^2-\omega_0{}^2)}$. Differentiating 3.8 we get

$$\dot{x}(t)=\alpha/\omega[exp(-\beta t)sinh\omega t] \qquad 3.9$$

The maximum value of 3.8 is given by

$$x_m=\alpha/\omega_0{}^2 \qquad 3.10$$

The maximum value of 3.9 is given by

$$\dot{x}_m=\omega_0 exp\{-(\beta/2\omega)ln[(\beta+\omega)/(\beta-\omega)]\}x_m \qquad 3.11$$

Since ω_0 and β are constants peak velocity $\dot{x}_m$ is directly proportional to amplitude x_m.